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An Address.[1]

By B. W. BUTTSWORTH,

Retiring President, Western Australian Branch of the British Medical Association.

THE Western Australian Branch of the British Medical Association was formed in 1899 under the presidency of Dr. J. A. Waylen. Its purpose today, as it was then, is to further the scientific learning, to guide the public relations and to uphold the ethical traditions of the profession.

This address will note some significant changes in Branch organization during the past year, will record the virtual attainment of the Medical School, which was the major achievement of the year, and will then consider some problems which in my view are important to the profession.

It became apparent that the affairs of the Association required the attention of a full-time secretary. Mr. Neilson Hancock had been part-time secretary for eighteen years and Mr. Hugh Hancock for ten years. The Branch is indebted to them for capable service. From over 30 highly qualified applicants, Mr. R. G. Hayward was selected. He has taken up his task with ability and enthusiasm. His increasing knowledge of British Medical Association affairs will greatly assist future Councils, and the growth of the British Medical Agency Company, already apparent, opens up a prospect of a very substantial income if all members would use the Agency. During the past six months the number insuring cars or houses through the Agency has increased to 50% of membership. However, only 90 members or 15% purchased through it in the past eight months. Increased use of this facility would enable Council speedily to achieve the amenities such as British Medical Association club rooms, reduction in annual subscriptions and many other things which members request. Council gets a lot of criticism from members. I am now criticizing members, that Council has in the British Medical Agency Company created a means of obtaining great benefits for the profession without cost to any member, yet the majority through indifference or indolence will not bother to ring the secretary when insuring or making a purchase for surgery or home.

During the year we transferred the British Medical Association Council rooms and office from Shell House to our own premises in Kings Park Road. Increasing rentals and parking difficulties made this change necessary. The project of a new building at 8 Kings Park Road will be the subject of a separate report. I only comment that the venture will succeed just as surely as this great State of ours continues to grow in population and wealth.

I turn from domestic affairs to a subject which is a source of pleasure and pride to every member of the

[1] Read at the annual meeting of the Western Australian Branch of the British Medical Association on March 17, 1956.

Branch—the attainment of a medical school in the University of Western Australia is of historic importance in the annals of medicine. It therefore seems appropriate at this moment to place in our records a brief story of persistent efforts which, after many disappointments and frustrations, finally achieved success.

In 1942 the Royal Commissioner, Mr. Justice Wolff, strongly recommended the establishment of a medical school. He estimated the minimum cost of buildings to be £100,000, and suggested that the Government set aside an annual sum over ten years for this service. In September, 1943, the Senate of the University appointed a Medical School Foundation Committee. As a result of this committee's recommendations, a deputation appointed by the Senate waited on the then Premier, Mr. F. J. Wise, in May, 1946. Mr. Wise invited Professor P. MacCallum, later Sir Peter MacCallum, of the University of Melbourne, to report on the matter. In his report in October, 1946, Professor MacCallum stated that he was convinced that a medical school should be established, but stressed the need to maintain the best university standards. The Senate informed the Premier that an early decision was necessary if teaching was to commence in 1948. Early in 1947 the Federal Government agreed to find £125,000 towards the cost of buildings.

Mr. (now Sir Ross) McLarty succeeded Mr. Wise as Premier, and stated that his Government would commence the medical school as soon as it was practicable. Subsequently he stated that the housing shortage made it necessary to defer the project. In 1948 the Vice-Chancellor again discussed the matter with the Premier and was informed that the housing position precluded any action for two years.

In November, 1948, the Senate approved the creation of the Advisory Board in Medicine, which included a number of members of the British Medical Association. This Advisory Board, in effect, replaced the Medical School Foundation Committee. On the recommendation of the Advisory Board, the Senate in May, 1949, submitted to the Premier a plan for immediate implementation. The Premier again announced that action was deferred.

Early in 1950 the proposal to establish a chair in medicine or pathology, with the professor initially confining his duties to post-graduate teaching, was accepted by the Government, but was not implemented by the University. At this time the Government agreed to the establishment of a Post-Graduate Research Unit at Royal Perth Hospital.

In 1951 on the recommendation of the Vice-Chancellor, Dr. G. A. Currie, the Senate launched a public appeal, the Sir James Mitchell Appeal, for funds towards a medical school. This appeal raised £17,589, to which was added £39,000 given by various bequests. At the same time, the Senate requested the Premier to approach the Federal Government for an increase in its offer of £125,000. This the Premier declined to do.

In 1952 an offer by the Senate to endeavour to raise £250,000 if the Federal Government were approached for a like sum was declined. Early in 1953 a new Government was elected, and the Premier, the Honourable A. R. G Hawke, told a deputation from the Senate that his Government would endeavour to find the capital cost of starting the medical school on a pound-for-pound basis with the Federal Government. It was now considered that the total capital cost would be £500,000 or £700,000. In December, 1953, the Premier informed the Senate that the Prime Minister, besides being unable to accede to the request for an increase in the original offer, had stated that the original offer of £125,000 had been cancelled. The prospect was dim, and became darker when in 1954 the Premier was informed that, from the beginning of 1956, the University of Adelaide would be unable to admit any new Western Australian students to the clinical school.

However, in June, 1953, a new approach in thought on the capital cost had been suggested in a report to the Senate by the Vice-Chancellor, Mr. S. L. Prescott. He had been discussing the medical school with government and hospital authorities, and it appeared possible that a pre-clinical school could be established in two army huts at the University, and that the older section of the Royal Perth Hospital could be converted for use in the clinical school. The idea, which rapidly found favour, was that while teaching, academic standards and equipment were to be well endowed and of the highest order, the buildings in the beginning were to be of a modest nature. The ultimate building in brick or marble would be better suited to the Faculty if designed after a few years of growth.

In May, 1954, the Advisory Board in Medicine considered a report by Dr. N. D. Crosby, reader and head of the department of physiology, outlining an interim plan to establish the school temporarily using modest buildings. This, with a summary by the Vice-Chancellor of the capital and annual costs, was submitted to the Premier in June, 1954; but by the end of November, 1954, no decision had been made by the Government.

During these months the Council of the British Medical Association became increasingly concerned at the approaching crisis in medical education. Members of the Advisory Board in Medicine, all senior members of the profession, agreed that every possible approach had been made to the Premier and counselled patient waiting for an expected announcement.

Public feeling was now stirring in the matter. A deputation from the Rotary Club of Perth assured the Premier of the support of the Rotary movement. The Premier told the deputation that he would give his answer later. Finally, at a meeting of representatives of the Branch Council with the members of the Advisory Board in Medicine, it was decided that a deputation from the British Medical Association should wait on the Premier. Your Council approached the Press, which agreed that considerable newspaper publicity would follow the deputation if necessary.

The deputation waited on the Premier on January 19, 1955. The spokesman, Mr. J. P. Ainslie, presented a very strong case and assured the Premier of the support of the whole profession. To the great relief and joy of all, the Premier announced that the State Government would meet half the initial cost and all the administrative cost of a medical school. He appointed a committee to advise the Government of an estimate of costs and requirements. This committee worked strenuously and reported to the Premier on April 4, 1955. On April 6 the Government announced that it had accepted the scheme and would meet half the cost of £300,000 initial expenditure and the whole of the annual cost of £100,000. The aim was to start clinical teaching in 1957 and pre-clinical teaching in 1958.

The task then was to raise a minimum of £150,000 by public subscription, and for this purpose the Senate of the University appointed an appeal committee, which was wise in seeking and fortunate in obtaining the services of the Administrator of the Royal Perth Hospital, Mr. J. Griffiths, and the Vice-Chancellor, Mr. S. L. Prescott, as honorary directors. The committee set itself the task of raising a minimum of £150,000 for capital requirements and a further £250,000 to provide funds which could be expended during the first five or ten years of the life of the medical school to ensure the recruitment of the best available professional staff and the provision of the very best facilities for research during that period. Many people doubted if £150,000 could be raised, and to most the total of £400,000 seemed impossible of attainment by a State with a population of 650,000 and relatively limited resources. In Western Australia a charitable appeal which raised £50,000 had hitherto been regarded as highly successful. The public of Western Australia responded to the challenge, and to the present date have subscribed £523,000, which includes £17,589 from the Sir James Mitchell Fund. To this fund may be added certain special benefactions amounting to £39,000, giving a total of £562,000. It was announced during the appeal that the balance over £400,000 would form the nucleus of a permanent endowment fund for research.

This was a truly remarkable response by the people of Western Australia, and the combining of all sections of

the community in a common cause should result in a school of such standing as will attract further liberal endowment for research by wealthy overseas institutions.

It was particularly pleasing to find how many were inspired to work and give by a feeling of gratitude for services rendered by the medical profession. In these days, the fact that a large majority of the profession is efficient and devoted is not news; it is the occasional error or neglect which makes the headlines. This assurance of the standing of the profession in the esteem of the public was therefore very welcome.

The members of the Branch responded generously in service to the appeal and in giving. There are 578 members in the State, of whom 72 are resident medical officers and 110 are members of the salaried services. This membership confounded the Jeremiahs (who predicted a maximum giving of £5000) and proved its devotion to the future of medicine by contributing £40,000. It was important, too, that they donated early and thus set a standard for others. When business firms were approached for donations, the first question asked was frequently: "What is the medical profession giving?" It is certain that an attempt to mention the names of all those who assisted would result in injustice to a number of our members. I therefore, on behalf of the Association, express deep gratitude to those who through the years have laboured in the Senate and Advisory Board in Medicine, and to those who assisted with the appeal and donated to it. The response by the profession to this appeal will, I believe, endure as one of the finest moments in the history of medicine in this State. It is my pleasing duty to convey to you the appreciation and thanks of the Senate of the University and of the Appeal Committee.

I now wish to speak of one or two problems, and, in doing so, will state my own views and not necessarily those of Council.

In the past few months there has been adverse publicity in the Press regarding excessive visiting under the National Health Scheme and the Pensioner Medical Service. We are assured by members of the Committees of Inquiry in this State that the fault exists here, but is not widespread and that they expect to bring it further under control. In passing, these men merit the thanks and support of the whole profession for undertaking a very onerous task. However, even if deliberate over-visiting is excluded completely, I consider that the Federal Government and the insuring bodies will continue to be alarmed by the astronomical costs of any medical service which is totally or largely free. This alarming rise in costs beyond all estimates is common to all social services throughout Great Britain, America and Europe. The big problem in the medical service is not one of over-visiting of the patient by the doctor, but one of over-consulting of the doctor by the patient. This over-consulting for early symptoms is natural and understandable. It is ideal if the country can afford it. It avoids the occasional tragedy which arises from late consulting. Sir Earle Page, when Minister for Health, emphasized how much it saved in hospitalization and loss of working time. It would be interesting to have an actuary report on the debits and credits of this matter. I consider that this problem will increase, and wish to say that it is quite unjust and unwarranted that the medical men of this country should be held entirely responsible for it. It is still true that it is the duty of each of us, as far as it lies in his power, to make the scheme succeed. It is also in his interest. If this scheme fails, far worse may befall.

The thought of the cost of social services brings me to another matter which, I think, is worthy of consideration, the economic position of the medical man himself. This may seem inappropriate to a presidential address, but I suggest that a doctor will give his best service to the community if he is not harassed by a burden of unreasonable financial concern. It appears that social services will increase; therefore taxation will increase also. At present a young doctor, having studied for six years at considerable financial sacrifice to himself or relatives, sets up practice and suddenly earns an income. At the end of his first year of practice he has to pay tax on his first year's income and provisional tax on his expected second year's income. He has to live, and probably to buy a house and car and keep a wife. Later he has to maintain a certain standard of living and educate his children. If he does manage to save a little, he has neither the time nor the training to invest wisely. He either puts his money into bonds and finds later that because of inflation his bond has lost value, or he puts it into shares in an insecure company. Yet he has, by the time he is forced by increasing age to retire from practice, to own a house and car and save an additional £15,000, which at present rates of interest would give him a standard of living equal to one on the pension. I therefore suggest that the peculiar circumstances of his commencing practice demand an attempt to make the incidence of provisional tax more gradual. The problem of sound investment of his savings would be solved if the British Medical Agency Company set up an investment trust. The practitioner would simply pay his savings into this trust knowing that it would be soundly invested. I commend this thought to the directors of the British Medical Agency Company. The problem of provision for old age obviously requires the amendment of the *Income Tax Assessment Act* to allow superannuation of self-employed persons as a taxation deduction. In league with the legal fraternity and others, the British Medical Association is endeavouring to have the Federal Government make this amendment. I suggest that each of you should bring this matter prominently before your Federal member and that a deputation should meet members of both parties when they are next in Perth.

During the year, certain difficulties arose in the matter of Press publicity. Council has recently acted with a view to more harmonious working of this difficult department in our State and supported the retaining of a trained public relations officer by the Federal Council. Although this was deferred at a recent meeting of Federal Council, efforts to secure this wise provision will continue.

It is time this meandering address came to a conclusion. I thank all members of Council and committees, Dr. Colin Anderson, Mr. R. G. Hayward and Miss W. Lunnon, and all members of the profession for their cooperation and tolerance during the past year.

DR. EDITH PECHEY, ONE OF THE FIRST WOMEN IN MEDICINE.[1]

By S. DEVENISH MEARES,
Sydney.

IF, as doctors, we are interested in the struggles of the first women to enter the profession, as Australians we should have a special regard for one of them. This was Mary Edith Pechey, who throughout her life had many links with Australia. William Pechey, a Baptist minister, married Sarah Rotton at Bath, England, on July 31 in the year 1837. Sarah was then thirty-one years old, and in 1846, when she gave birth to her daughter Edith, she already had three sons and three other daughters. How the Reverend William Pechey, from his stipend, was able to find bread for their mouths and clothes for their backs is a matter of wonder. How, too, Mrs. Pechey found time to look after seven children under nine years of age as well as carry out her many duties as minister's wife is hard for us in these complex times to imagine. No doubt she had help from her sisters and perhaps also from the people of the parish. Mrs. Pechey was accustomed to a house crowded with children. Her mother had died giving birth to her thirteenth child after seventeen years of marriage.

Edith Pechey was the youngest child and she must have soon found that she must struggle for existence. However,

[1] Read at a meeting of the Section of History of Medicine, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

judging by her later behaviour, it is clear that she soon learned that more was to be won by sweet reasonableness than by aggression. That the family thoughts and conversation were not taken up merely with domestic and local matters is certain. Often the talk was of overseas, for when Edith was born two of her aunts and two of her uncles were in America with their families. Henry, another uncle, had married in Australia Lorna Jane McPherson (a great-aunt of Dr. John McPherson, one-time lecturer in medical jurisprudence at the University of Sydney). Henry's daughter Caroline was to marry Henry Keightley and encounter the bushranger Ben Hall.

This pioneering self-reliant spirit was also infused into the brothers and sisters of Edith Pechey. One of the brothers died on the way to America, and two brothers married in Australia. A sister Jane married Dr. Alfred Shewen, of Sydney, honorary physician to the Royal Prince Alfred Hospital. It was Dr. Shewen who, at the Intercolonial Medical Congress in Sydney in 1892, read a paper entitled "Death under Anæsthesia".

Let us now return to the year 1863 and the city of Bath, where the minister's wife is busy with her family. Her daughter Edith was now seventeen years old. The mail arrived from Australia, and with it no doubt a copy of the *Bathurst Times* of October 28. All would be excitement as they read of the attack of Ben Hall and his gang on Dunn's Plains Homestead, the home of Edith's cousin Caroline Keightley. Caroline's husband was a magistrate and, although caught without his main arms, returned the bushrangers' fire with a shot gun. During the fight in defence of his wife and baby Keightley shot Mick Burke, one of the gang, in the stomach. Dr. Pechey, another cousin of Edith, who was visiting Australia, was staying with Henry Keightley and took part in the fight. Eventually, ammunition becoming low and having received a promise that the women would not be molested, Keightley and Dr. Pechey surrendered. Dr. Pechey was called on to attend Mick Burke, who died before the end of the day.

Ben Hall demanded a ransom of £500 for the life of Henry Keightley. This sum was the same amount that was on his own head. Caroline, in the early hours, with Dr. Pechey drove to Bathurst to obtain the money from her father, Henry Rotton, M.L.A. In view of the fact that 1955 is the centenary of Cobb and Company, it is interesting to recall that Henry Rotton once controlled the Sydney-Bathurst mail coaches. The money was paid in marked notes, and this later helped towards the rounding up of the whole gang.

In the year 1869, when she was twenty-three years old, and while acting as a governess to some children in a private home, Edith Pechey decided that she wished to become a doctor. Elizabeth Blackwell in 1850 had been the first woman in America to become a doctor, and in 1865 Elizabeth Garrett passed the final examination of the Society of Apothecaries, London, and became the first woman in England legally qualified to practise in medicine. The Society of Apothecaries later altered the regulations by making a rule that a certificate of instruction should be presented from a recognized medical school. A letter from a Mrs. Butler to Miss Sophia Jex-Blake, who in 1865 had unsuccessfully applied to the University of Cambridge to study medicine, contained the following: ". . . Have you seen Miss Pechey? She did not seem to me very clever but very steady and nice, a silent quiet woman."

It was not for any selfish reasons that Edith Pechey wished to do the medical course, nor did she underestimate the difficulties ahead of her. She wrote:

> Before deciding finally to enter the medical profession, I should like to feel sure of success—not on my own account, but I feel that failure now would do harm to the cause, and that it is well that at least the first few women who offer themselves as candidates should stand above the average of men in their examinations. Do you think anything more is requisite to ensure success than moderate abilities and a good share of perseverence?
>
> I believe I may lay claim to these, together with a real love of the subjects of study, but as regards any thorough knowledge of those subjects at present, I fear I am as deficient as most. I am afraid I should not without a good deal of previous study be able to pass the preliminary exam, you mention, as my knowledge of Latin is small and of Euclid still less. Still, if no very extensive knowledge of these is required (and doctors generally seem to know very little of them) I could perhaps be ready by the next exam.

About this time Miss Jex-Blake wrote to the Senate of the University of Edinburgh offering, on behalf of her fellow potential students and herself, to guarantee whatever minimum fee the Faculty might fix as a remuneration for the separate classes for women. When she heard of this Edith wrote to Sophia Jex-Blake: "I shall be sorry if my means will not allow me to take a full share of the expenses but I am afraid I shall not be able to afford more than double the usual fees for a man." Sophia wondered whether so pretty a girl as Miss Pechey could have nerve enough to study medicine and said she had been informed that the pretty girl was as "calm as an ox".

On October 10, 1869, Edith lunched with Sophia, who was six years her senior, and a week later Sophia wrote about Edith to a friend:

> I think her strong, ready handed with faculty, great ability, resolution, judgment, great calmness and quiet manner and action, and probably great strength of feeling, good taste, good manner, very pleasant face; considerable sense of humour; lots of energy and interest in things. In fine as good an ally and companion as could well be had.

In a later letter to Dr. Lucy Sewell, of America, she says: "Miss Pechey is very nice and very clever,—you will like her very much, and she is such excellent company."

Eventually in 1869 they were, with three other heroic women, permitted to start their medical course. When the winter session came to an end in March, 1870, it was found that in chemistry Miss Pechey stood third in the entire list of candidates. She was really top of the first-year students, as the two men above her on the list had attended the class the previous year. Her success qualified her for a Hope Scholarship, which amongst other things would have entitled her to the free use of the college laboratory. This would have put her in a class with male students, so the prize was given to the man who stood beneath her on the list.

The professor, explaining this injustice, said: "Having studied at a different hour, she was not a member of the chemistry class." At the same time he awarded her the official bronze medal of the university. However, she could only lay claim to this award as a member of the chemistry class. This injustice soon became public, and Miss Pechey became the recipient of an amount of condolence that was embarrassing. The editor of *The British Medical Journal*, April 16, 1870, wrote: "The University has done no less an injustice to itself than to one of its most distinguished students." Protests were made in all the leading papers, including *Punch*. During all this turmoil the pretty girl, who was as calm as an ox and who was on holiday in the country, wrote to her friend Sophia: "I am very vexed but it is no use worrying,—at least so the nightingale tells me. She sang two hours at my bedroom window last night, and said all sorts of pretty things."

This opposition and hostility to women medical students continued. In the year 1870 mud and egg throwing was coupled with vile abuse from groups of male students. Twelve judges of the Inner House in Edinburgh decided that the University had exceeded its powers in 1869. The examiners of the Royal College of Surgeons of England resigned and the council failed to appoint others to examine the women candidates. The presidents of both the Royal College of Surgeons of Edinburgh and the Royal College of Physicians of Edinburgh refused to take the chair when women were amongst the prize-winners.

In the year 1874 there was still no hospital open for the clinical training of women students. Three of the students, Miss Jex-Blake, Mrs. Isabel Thorne, and Miss Pechey, together with Professor Huxley, F.R.S., and others, met at the house of Dr. Anstie at 69 Wimpole Street on August 22,

1874. They formed the provisional council which founded the London School of Medicine for Women, and in October of that year 14 students were enrolled for non-clinical studies. It is interesting to note that Alfred Shewen, M.D. (London), who was mentioned earlier, was at one time lecturer in forensic medicine at this school. In 1877 the school was linked with the Royal Free Hospital for clinical teaching.

FIGURE I.

Mary Edith Pechey-Phipson, M.D. (From a painting in the Cama Hospital, Bombay.)

By the year 1876, seven years after the women had started the medical course, there were still only the names of two women on the medical register. In September, 1876, Miss Pechey and Miss Shove went to Ireland. There they persuaded the Queen's University and the King's and Queen's College of Physicians to allow those women who had studied in Edinburgh to sit for their final examination. Mrs. Isabel Thorne then wrote: "Miss Pechey was a loyal and stimulating comrade throughout, disarming opponents by personal charm, intelligence and humour." All the women students succeeded in their final medical examinations in 1877, and so were registered by the General Medical Council of Great Britain and Ireland. In the same year Miss Pechey took the doctorate in medicine of the University of Berne with a thesis on "The Constitutional Causes of Uterine Catarrh". No English university would at that time grant a degree to women.

After graduating, Edith Pechey obtained a post as house surgeon in the Hospital for Women, Birmingham, under Lawson Tait, who was the pioneer of abdominal and pelvic surgery. These two pioneers must surely have understood each other. The training in abdominal surgery proved a great asset in her later practice. Subsequently, for six years she was in private practice in Leeds.

It was while doing further post-graduate surgical study in Vienna in 1883 that she received word of her appointment by the Medical Women of India Fund to be head physician at the dispensary which was to be opened at Bombay. Edith Pechey arrived in Bombay on December 12, 1883, and took charge of the dispensary. By her work she raised the whole standard of nursing in India in the face of strong, ancient prejudices. In August, 1886, a Parsee gentleman, Mr. Cama, founded a hospital in Bombay bearing his name, for the care of women and children, with Dr. Pechey in charge. To quote from "Medical Women in India":

> The Medical Women of India Fund was fortunate in its choice, for Dr. Pechey was not only fitted by talent, education and experience to take a foremost place among the members of the medical profession in Bombay, but she was able to take part in the social life of the city, and so secured a greater recognition of the valuable work done in the Cama Hospital. She was also a great public speaker and soon became widely known and appreciated by all classes.

Later the services of Dr. Pechey and of her assistant, Dr. Charlotte Ellaby, were taken over by the Indian Government. In addition she had a large private practice, both European and Indian. Often she was called to zenanas of Indian States to treat women in purdah and at times was rewarded with the gift of jewels. Also in her charge were the Ketwody and Alibless Hospitals in Bombay, and later she founded the Sanatorium for Women and Children at Nasik Road in the Deccan. Dr. Pechey, during her time in Bombay, undertook many activities outside her professional work, all aimed at improving the intellectual, political and social well-being of the women of India.

Notably, on October 11, 1890, Dr. Pechey gave an address on the evils of child marriage. This subject had been discussed from various points of view, religious, educational, political, moral and sentimental. It was left to a woman doctor to stress the physiological aspects. This was done by Dr. Edith Pechey with great ability, tact and good feeling. The address was published in many languages throughout India and was quoted extensively by many Indian reformers. There is no doubt that her testimony did much to alter the Indian law. Dr. Pechey also supported an Indian girl, Rukhmabai of Rajkot, who went to England, against all precepts, and qualified in medicine in Britain. On June 23, 1886, Rukhmabai wrote to Dr. Edith Pechey:

> My Dear Miss Pechey,
>
> I hereby return one of your books (The Roman Singer) with many thanks. I looked it all over just enough to know the purport of the story which I found contains nothing but mere love matters. I shall return the other book (Medical Women) in a few days.

Edith Pechey, at the age of forty-three years, in 1889 married an Englishman, Herbert Musgrave Phipson. Herbert Phipson was a wealthy wine merchant in Bombay, a man of great charm, and interested in scientific pursuits, honorary secretary of the Natural History Society of Bombay, and a member of the Lady Dufferin Trust. They entertained lavishly, and Mrs. Pechey-Phipson's great social gifts, humour and powers of conversation made her a brilliant hostess. May Thorne, daughter of Isabel Thorne, who you will remember was an important member of the "Seven against Edinburgh", stayed with the Phipsons for a few months before she took up the study of medicine.

Edith Pechey-Phipson was appointed to the Senate of Bombay University, a unique honour for a woman. The climate and strenuous work took their toll of her health. In 1893 she was obliged to resign her hospital appointments and return to Britain. During the plague years of 1896 to 1898 she returned to India to help, and was the first to organize and take part in house-to-house visitation, a dangerous and arduous undertaking.

On June 13, 1891, an account of her work and a photograph were published in *The Town and Country Journal*, Sydney. In 1905, with her husband, she left India and spent more than six months in Australia and New Zealand.

Edith Pechey-Phipson was a notable suffragette. By the year 1894 the women of Wyoming (United States of America), Finland, New Zealand and South Australia had been given the vote. With Federation all Australian women were enfranchised. In Britain it was not until 1918 that an Act was passed giving the franchise to women who had reached the age of thirty. The women of Switzerland still have no vote.

In the year 1906, as the Leeds delegate, Dr. Pechey-Phipson attended the International Women's Suffrage Alliance held at Copenhagen. Her report to the Leeds Women's Suffrage Association on October 10, 1906, was published by The Women's Printing Society Limited, London. She says in the report: "When I was in Sydney I was invited to The Women's Club to meet some of those who had worked for the suffrage . . . ." Dr. Mary Booth, of Sydney, had been present on that occasion and later had visited the Cama Hospital and had stayed with the Phipsons in England. Continuing the report Dr. Pechey-Phipson says: "In four years the women of New South Wales have won reforms which the voteless women of Victoria (their neighbours) have asked for in vain for fifteen years." The writings of her friends and colleagues make it clear that she was a kindly generous woman of fine character, gregarious, with a sense of humour, withal a strong and picturesque personality. She was, however, throughout her life a pioneer and crusader, fighting for just causes, and overcoming the opposition of apparently overwhelming authority by her personal charm, intelligence and humour.

Edith Pechey was my maternal great-aunt. I cannot doubt an uncle of mine when he says that she had a fine character but was not soft, and had perhaps a little of male dominance about her. Nor can one do other than accept the picture, painted by the husband of one of my aunts, who met her for the first time in Perth while Edith Pechey and her husband were relaxing in a private sitting room in the Palace Hotel. The first thing he noticed were her large, black, low-heeled shoes, for she was sitting in an easy chair, with her feet on another chair, smoking a large cigar. At the same time her husband, slight, with red hair, stood beside the chair trimming one of her hats, an art at which he rather excelled.

In the year 1907 in London she underwent a successful operation by Dr. May Thorne, the daughter of her comrade of the battle, Isabel Thorne. Her health, however, had been undermined and Edith Pechey died peacefully in her sixty-third year at Folkestone on April 14, 1908.

Acknowledgement.

It is a pleasure to acknowledge the help given to me during the preparation of this paper by the late Dr. May Thorne, Mrs. H. M. Brandreth, librarian of the Royal Free Hospital School of Medicine, the librarian, Bombay University, Miss M. Rolleston, of the British Medical Association Library, Sydney, the librarians of the Mitchell Library, Sydney, Mr. R. I. Money, photographer, Sydney Hospital, and my own relatives.

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PESTICIDES AND THEIR TOXICOLOGY.[1]

By G. H. McQUEEN,
Adelaide.

THE demand for more food to feed the increasing population of the world has stimulated research into improved methods of food production and conservation. As a result, a wide variety of chemical substances, to prevent and control the living organisms which adversely affect food in its various phases, have been both discovered and used. Many of these substances, however, also affect human beings who come into contact with them. In this paper, after briefly describing their use in agriculture, I shall indicate the effect of some of them on human beings.

These chemical substances may become important, from the human being's point of view, during the manufacture or processing of the concentrated substance, during the dilution or mixing prior to its use, during the use or application, and following the consumption of food containing small amounts or residues that have remained after previous applications. In each case it is obvious that different groups of people may be involved and variable amounts of the substance may be introduced into the body to produce varying signs and symptoms of toxicity. The path of entry into the body may also vary in each instance. Generally the toxic effects can be divided into the acute, when a comparatively large single dose of the pesticide substance finds its way into the body, and the chronic, when small doses enter the body at frequent intervals over a long time.

Definitions.

In a discussion of pesticides and their toxicology it is necessary to define a number of terms commonly used.

"Pesticides" commonly include insecticides, fungicides, weedicides and rodenticides. They may be defined as products which will control any form of virus, plant or animal life. The term is, however, not usually applied to products that are used to control these forms of life when they are causing disease in human beings.

"Acute toxicity" is the term used when the toxicity produced is due to a single dose of the substance which is responsible for the resulting signs and symptoms of poisoning.

"Chronic toxicity" is the term used when the signs and symptoms produced are the result of repeated small doses over a long period.

"A lethal dose" is the amount of the poison which will cause the death of the organism, and the term "LD50" is applied to the dose which will kill 50% of a population to which it is given. Doses that are fatal for other proportions are referred to as LD, and added is the percentage killed. The dose is usually measured in milligrammes per kilogram of body weight of the animal under observation.

"Tolerance dose" is the safe upper limit fixed for a pesticide residue on foodstuff intended for human consumption.

[1] Read at a meeting of the Section of Public Health and Industrial Medicine, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

Methods of Grouping Pesticides.

Prior to the introduction of DDT a wide variety of substances, both organic and inorganic, were used as pesticides. These have been grouped together in various ways. One of these ways, depending on their mode of action against pests, groups them into (i) stomach poisons, (ii) contact poisons and (iii) fumigants. Another divides them into groups according to their particular use as herbicides, weed-killers, defoliants, fungicides, insecticides and ascaricides. Pesticides have also been subdivided into both comparatively safe and hazardous substances, according to their effect on human beings. There is probably no really satisfactory method of grouping the large variety and number of pesticides now in use, and it is not possible in this brief survey to discuss the toxicology of each one. There are more than 400 different brands of pesticides registered with the South Australian Agriculture Department.

I will therefore indicate in general outline the toxicology of the following groups: (i) a group of pesticides that owe their pesticidal effect to one or more of the metallic elements—arsenic, lead, mercury and fluorine; (ii) the halogenated organic compounds; these consist mainly of chlorinated hydrocarbons, but they also contain methyl bromide; (iii) phenol compounds; (iv) the organic phosphorus-containing compounds; these may be subdivided into systemic and non-systemic compounds; (v) others, including sodium fluoroacetate.

Metallic Pesticides.

Arsenic.

Fairhall states that one-half of the arsenic produced in the United States of America in 1945 was used in the preparation of agricultural insecticides. Its use in weed-killers, cattle dip and wood preservatives accounted for a substantial share of the balance. It is also extensively used in Australia; for example, every local authority in the Adelaide metropolitan area uses some form of arsenic preparation as a weed-killer on the footpaths.

In cases of acute poisoning with arsenical compounds, arsenic acts as a powerful protoplasmic poison, causing acute irritation or even destruction of the tissues. If taken by mouth, it produces inflammation of the intestinal tract, resulting in violent vomiting, diarrhœa and nervous collapse. Hæmolysis, with jaundice, oliguria and hæmaturia may be succeeded by complete suppression of urine. If death does not occur, sensory nervous disturbances develop, and these are followed by motor paralyses, loss of hair, nail deformities and skin lesions.

In cases of chronic poisoning, particularly when the arsenical is in the form of a dust or mist, the skin and mucous membranes are irritated and the subject experiences reddened eyes, conjunctivitis, sore throat, hoarseness, coryza, perforation of the nasal septum, skin rashes, ulcers, keratoses and epitheliomata. Slow absorption through the skin occurs and causes bronzing. Loss of hair and trophic changes in the nails are common. Hallucinations, aphasia and peripheral nerve lesions may also occur. Lead arsenate, the commonly used arsenic-containing pesticide, is soluble in the digestive juices with the liberation of both lead and arsenic. These may produce signs and symptoms of chronic poisoning by both. In cases of chronic poisoning, arsenic is excreted in the urine in amounts greater than the normal value of 0·008 to 0·05 milligramme per litre.

Lead.

Lead is a protoplasmic poison, and in cases of acute poisoning following the ingestion of lead salts, the signs and symptoms are those of acute irritation and destruction of the cells lining the alimentary canal. If death follows, it can be attributed entirely to this damage. When used as a pesticide, lead is usually combined with arsenic as lead arsenate. Lead compounds in common use as pesticides may enter the body either by inhalation or by ingestion.

In chronic poisoning, absorbed lead is carried to all parts of the body and causes extensive cellular damage. It is excreted from the liver and kidneys, and at the same time it is deposited in the bony skeleton. This cell damage manifests itself in the production of a typical syndrome of signs and symptoms associated with lead absorption and lead poisoning. These include intestinal colic, a lead line on the gums, basophilic stippling of the red cells, increased urinary excretion of lead, muscular paralyses, and anæmia. Goldblatt considers that the anæmia is due to both incomplete hæmoglobination of the red cells and an increased rate of destruction of red cells containing lead by the reticulo-endothelial cells in the spleen and elsewhere.

Mercury.

Mercuric salts and compounds, particularly the organic compounds, are extensively used as fungicides in the treatment of seeds, the purpose being to kill the spores of plant fungus diseases. Mercury is of interest because it volatilizes even at low temperatures. This property assists its fungicidal action, but is a disadvantage from the human being's viewpoint. The air in rooms where seed which has been treated with a mercury compound is stored will contain a proportion of the mercury compound. This metal can enter the body from the respiratory tract and the intestinal tract and through the skin. It is a severe protoplasmic poison and causes irritation and severe injury or death to living cells. In cases of acute poisoning, if it is swallowed, corrosion may occur in the mouth, throat, stomach and intestines, with severe vomiting of blood-stained material succeeded by profuse diarrhœa with watery, blood-stained motions. Death may follow severe shock and collapse. If the patient survives the initial effects of an acute toxic dose, he usually develops severe colitis with diarrhœa, melæna and abdominal colic. After one or two days severe nephrosis develops, with first an increased and then a decreased output of urine which contains albumin, blood and casts. This condition may be followed by anuria and subsequently uræmia, with convulsions, coma and collapse. Repeated small doses of mercury eventually produce a type of condition portrayed by the Mad Hatter in "Alice in Wonderland". This condition is often found in persons handling mercuric compounds in industry. The main signs and symptoms are an intention tremor, a characteristic psychological disturbance known as erythrism (a peculiar form of timidity), pallor and stomatitis. This last manifests itself as tender inflamed gums, pyorrhœa and sometimes excessive salivation. A urinary excretion of more than 0·25 milligramme of mercury per litre of urine suggests over-exposure to mercury. The organic compounds of mercury are probably now more frequently used as pesticides than are the inorganic compounds. In 1940 Hunter reported four cases of mercury poisoning in persons working with methyl mercury compounds. In these patients the signs and symptoms were entirely nervous in origin. Paræsthesiæ of the extremities occurred, and later there were tremor, dysarthria, ataxia, non-coordinated movement and restriction of the visual field. Severely affected persons may be permanently crippled and unable either to stand or to speak intelligibly. A similar case occurred in Adelaide in 1952.

A person became ill while using "Ceresan" (Bayer) as a seed dressing. This substance contained aryl and alkoxy ethyl mercuric compounds. The symptoms of poisoning were headache, vomiting, "pins and needles" in the hands, abdominal pain and a "slimy" taste in the mouth. Signs were those of meningism, pallor of the optic disc and an apathetic mental state. A month after his admission to hospital, the patient's urine contained 0·05 milligramme of mercury per litre.

Sodium fluoroacetate is a fluorine-containing compound which will be considered later.

Halogenated Organic Compounds.

Chlorinated hydrocarbons are the largest group among the halogenated organic compounds. They include DDT and a large number of related organic compounds with

similar toxic effects. During the early years of the second World War there was an intense search for efficient, easily produced insecticides to meet the greatly increased demand, both to protect armies from insect-carried diseases and as substitutes for insecticides made unavailable on account of the war.

DDT.

DDT (dichloro-diphenyl-trichlorethane) was the first of the new synthetic pesticides. It was originally produced by the Geigy Company in Basle, Switzerland, in 1938. It was extensively used during the later years of the second World War, mainly as an insecticide to control mosquitoes, body lice and flies, the vectors of malaria, typhus and dysentery respectively. With the advent of peace, DDT became available for civilian use and experimentation. In addition to its use in controlling the vectors of disease, it was found to be useful in controlling a wide range of agricultural pests. As it was not equally effective against all such pests, the search for more effective pesticides was continued and still continues today. DDT is a tasteless white powder with a fruity odour. It has many isomers, the para prime being the most active insecticide. It is both a contact and a stomach poison for insects. In man and the higher animals it is absorbed through the skin, from inhaled air in the lungs, and from the intestinal contents. The amount absorbed varies with the solvent used. It is increased and more rapid when the solvent is either an oil or a lipoid solvent. In the body some is stored unchanged in the fat and some is broken down to other substances. Two of these have been identified. One, an acetic acid derivative of DDT, is excreted in the urine, and the other, a dehydrochlorinated derivative, is stored in the fat. The output of the acetic acid derivative in the urine is related to the DDT intake and can be used as an indication of DDT intoxication.

The toxic effects are mainly due to a poisoning of the cells of the central nervous system producing hyperexcitability, tremors, spastic and flaccid paralysis and convulsions. Damage to the liver and kidney cells and fatty degeneration of the myocardium have also been noted in cases of acute poisoning. In chronic poisoning there are loss of appetite, gastric discomfort, abdominal pain, loss of weight, mild anæmia, muscular weakness and tremors that may develop into convulsions followed by coma and death. Absorption through the skin may result in both diminished reflexes and patchy peripheral anæsthesia, in addition to the above. These effects, in varying degrees, are produced by other members of this group of insecticides. They include aldrin, dieldren, isodrin, endrin, BHC or gammexane, lindane (the γ isomer of gammexane), chlordane, heptachlor, and methoxychlor.

Methyl Bromide.

Methyl bromide is a halogenated, organic substance and is used extensively as a fumigant. It is also used both as a refrigerant and as a fire extinguisher. It is a stable chemical which boils at 40° F. It has a vapour density of 3·27. It has exceptional powers of penetration which make it valuable as a fumigant, and it does not leave a dangerous residue or impart an objectionable odour or taste to foodstuffs. Methyl bromide is an acute irritant and protoplasmic poison. Inhalation of a sufficient amount causes irritation of the respiratory tract and results in pulmonary œdema and death within a few hours. Lower concentrations cause nerve cell irritation and damage, with resulting headache, visual disturbances, nausea, vomiting, listlessness, vertigo and tremor, and these are succeeded by convulsions, pulmonary œdema, cyanosis and death. In patients with milder, non-fatal poisoning, nearly every type of nervous disturbance has been reported. When applied to the skin, methyl bromide is extremely irritant and may produce severe burns.

Phenol Compounds.

The principal compounds in this group are pentachlorophenol and its salts, the dinitrophenols and the dinitrocresols.

Pentachlorophenol.

Pentachlorophenol is a greyish, flaky, chemically stable substance. It has a low vapour pressure and it is only slightly soluble in water. Its low vapour pressure ensures its permanency. When heated, it gives off irritating, pungent fumes. It is very toxic to many types of organisms. These properties make it an efficient preservative of timber, glues, gums, hemp, jute and textiles generally. It is also used as a weedicide in Queensland in the cultivation of pineapples. Human beings can absorb it through the skin, the respiratory tract and the digestive tract. Its toxicity for a number of animal species has been determined. The LD50 for rats, following oral administration, is 78 milligrammes per kilogram of body weight. Test animals developed acute toxæmia, hyperpyrexia, hyperglycæmia, increased peristalsis, muscle weakness and asphyxial convulsions.

In humans irritation of the eyes and the upper part of the respiratory tract has been noted a few hours after exposure to a large amount of the chemical. Neuralgic pains in the legs, acne and dermatitis have been attributed to chronic exposure.

Dinitro-orthocresol.

Dinitro-orthocresol is a yellow crystalline substance with a melting point of 86° C. It is only slightly soluble in water. It is used as a weed-killer, fungicide and ovicide. The sodium salt is used to control broad-leafed weeds in cereal, pea, maize, flax, garlic and onion crops. It can be absorbed by humans through the skin, lungs and intestinal tract. The signs and symptoms of toxicity produced are those of excessive stimulation and increase in the general body metabolism. These are excessive sweating, thirst, increased respiratory rate, hyperpyrexia and fatigue. Death, if it occurs, is due to hyperpyrexia or heat stroke. In less acute and in chronic poisoning, loss of weight occurs; the affected person usually feels particularly fit, he has an increased basal metabolic rate, and his face, hands and hair are usually stained a persistent yellow. Other conditions attributed to dinitro-orthocresol are papular dermatitis, acute skin burns, nasal irritation, cataract of the eye and peripheral neuritis in the feet and legs.

Biochemical investigation has indicated that dinitrophenol, pentachlorophenol and dinitro-orthocresol interfere with the phosphorylating processes of carbohydrate metabolism in such a way that both the oxygen demand and the metabolic rate of the body are increased. Deaths from dinitro-orthocresol poisoning occur more frequently when the climatic conditions are hot and humid. Under these conditions the body experiences increasing difficulty in dispersing heat, and hyperpyrexia and heat stroke result more quickly.

Organic Compounds of Phosphorus.

The organic compounds of phosphorus include a group of pesticides with an insecticidal action similar to that of nicotine. They were first discovered in Germany in 1939 and were developed there during the second World War as insecticides to replace nicotine. On account of their characteristic toxic effect on human beings, their use in chemical warfare was considered. After the war they were also manufactured in Great Britain and the United States of America. Further research resulted in the discovery of other related compounds still more toxic to human beings. Recently, however, compounds less toxic to humans and more toxic to insects have been developed. These compounds belong to a group of chemical substances that are enzyme inhibitors. Their toxic effects are due to their ability to inhibit or destroy cholinesterase, an enzyme involved in the transmission of nerve impulses across nerve junctions and nerve-muscle junctions. Normally acetyl-choline is released at these junctions during the passage of a nerve impulse. After the impulse has passed, the acetyl-choline is immediately hydrolysed to acetic acid and choline by the catalytic action of cholinesterase. When this action is completed the neuro-muscular system is ready to respond to the next impulse. If the acetyl-choline is not removed, as in the case of poisoning

by an organic phosphorus-containing compound, nerve impulses are blocked. They do not reach the muscles, which are then paralysed. When death follows poisoning by one of these compounds, it appears to be due to respiratory failure from paralysis both of the diaphragm and of the intercostal muscles. After a single sublethal dose, the cholinesterase activity returns to nearly normal within twenty-four hours, and the signs and symptoms disappear. After repeated doses, a cumulative toxic action develops and recovery is slow. This is probably due to the complete destruction of cholinesterase and the slow regeneration of enzyme protein.

TABLE I.

Halogenated Organic Compounds.

Compound.	L.D. 50[1] by Mouth in Rats.	L.D.[1] for Man.
D.D.T.	250	400
Aldrin	40 to 50	—
Dieldrin	50 to 60	—
Isodrin	40 to 50	—
Endrin	24 to 36	—
Benzene hexachloride ..	250	400
Lindane	125	—
Chlordane	500	—
Heptachlor	500	—
Methoxychlor	6000	—

[1] All values in milligrammes per kilogram of body weight.

The signs and symptoms of poisoning by an organic phosphorus-containing compound may be divided into the following three groups: those that resemble the effects of muscarine poisoning; those that resemble the effects of nicotine poisoning; and those due to its effect on the central nervous system.

Early signs and symptoms which resemble the effects of muscarine are usually anorexia and nausea followed by abdominal colic, vomiting, sweating, salivation and contraction of the pupils. These may in turn be succeeded by diarrhœa, involuntary defæcation and urination, pinpoint pupils, blurred vision, and respiratory œdema and failure.

Signs and symptoms which resemble the effects of nicotine are scattered, fine and coarse tremors followed by generalized coarse tremors and muscular weakness.

The signs and symptoms due to the effect upon the central nervous system are giddiness, restlessness, anxiety, headache and mental confusion followed by coma, in severe poisoning. Until recently it was thought that these substances did not produce chronic effects when sublethal doses were administered over a long period. However, Goldblatt has noted that a case of persistent paralysis due to "Parathion" was reported in Germany in 1951, and two similar cases occurred in England in 1953. Both cases were due to "Mipafox" (bismonoisopropyl-phosphorodiamidic fluoride). It is probable that the paralysis in these cases is due to demyelination in the spinal cord and brain.

All the organic phosphorus-containing compounds may enter the body through the respiratory tract from inhalation, through the intestinal tract from ingestion and through the skin and conjunctiva. The rates of absorption through these various routes may vary with the different compounds. For example, the low oral toxicity of the newer organic phosphorus-containing compounds, "Chlorothion" and "Malathion", is attributed to poor absorption of these compounds from the intestinal tract.

Some of the organic phosphorus compounds are systemic insecticides. They are readily absorbed by a growing plant through the leaves and roots into the sap stream. This renders the whole plant insecticidal to insects sucking the sap and also toxic to human beings if any part of the plant or its fruit is eaten.

"Parathion."

"Parathion" (O,O-diethyl O-p-nitrophenyl thiophosphate) is probably the organic phosphate compound most used in Australia as a pesticide. It acts both as a contact and as a stomach poison as well as being an ovicide. It is stable in a neutral solution, but is rapidly hydrolysed in alkaline solutions.

HEPT.

HEPT (hexaethyl tetraphosphate) is used to a less degree than "Parathion". It acts as a fumigant as well as a contact and as a stomach poison. It is rapidly hydrolysed when mixed with water or exposed to moist air. The danger of poisoning by residual HEPT is therefore less than by residual "Parathion".

"Systox."

"Systox" (0, o-diethyl o-ethylmercaptoethyl thiophosphate) is a systemic organic phosphate compound in use in Australia. Its use is permitted in New South Wales on non-edible plants and on apple trees, if applied three weeks before harvesting. Its use is not permitted in South Australia. A recent development in the use of organic phosphorus-containing compounds has been the introduction of members of this group with high insecticidal activity and lower toxicity. "Malathion" and "Chlorothion" are both approximately 200 times less toxic to mammals than is "Parathion". "Meta-systox" (dimethyl ethyl mercapto ethyl thiophosphate) is approximately 20 times less toxic than "Parathion".

"Dipterex."

"Dipterex" (0,o-dimethyl-1-hydroxy-2, 2, 2-trichloroethyl phosphonate) is a recently introduced organic phosphorus-containing compound which has been shown to be highly effective in the control of house flies. Investigations into the toxicity of this compound indicate that it has a relatively low mammalian toxicity. It is 150 times less toxic than is "Parathion". This property is attributed to its rapid detoxification in animal tissues.

Sodium Fluoroacetate.

Sodium fluoroacetate, also known as Compound 1080, was originally introduced as a rat poison. It has caused considerable interest in Australia recently because of its use as a rabbit poison. Its use in America, where it was first manufactured in quantity, is strictly controlled. It was first synthesized in Ghent, Belgium, in 1896. In 1943 it was found to be the toxic substance in the South African poisonous plant *Dichapetalum cymosum*.

TABLE II.

Organic Phosphorus Compounds.

Compound.	L.D. 50[1] by Mouth in Rats.	L.D. 50[1] Intraperitoneally in Rats.	L.D.[1] for Man.
"Parathion"	15	7	125
HEPT	7	—	50
TEPP	2	0·65	—
"Paraoxon"	3·5	1·2	—
"Dimefox"	5	—	—
"Systox"	9	8	—
"Systox" (toxic isomer)	1·5	—	—
"Metasystox" (toxic isomer)	40	—	—
"Pestox" (O.M.P.A.) ..	10	8	8 to 22
"Malathion"	2860	750	—
"Chlorothion"	1500	750	—
"Dipterex"	450	225	—

[1] All values in milligrammes per kilogram of body weight.

It is a chemically stable, white, odourless and tasteless powder. It is very soluble in water and relatively insoluble in common organic solvents. Within the body, fluoroacetate becomes involved in the tricarboxylic acid cycle in the metabolism of carbohydrates to water and carbon dioxide. Fluoroacetate is transformed by the reactions of this cycle to fluorocitric acid, and this blocks the further progress of the cycle by inhibiting the action of aconitase. This last is one of the enzymes in the cycle involved in

the breaking down of citric acid. Consequently, there occurs an interruption to the energy supply to the nerve cells of the brain, sufficient to throw parts of it out of order and to produce convulsions. In addition, the mounting up of citric acid in the cells may interfere with the calcium concentration and also cause convulsions. Whatever the exact cause of the convulsions, it has been definitely shown that the action of fluoroacetate upon aconitase leads to a state of hyperexcitability of the neurons concerned.

The dose of fluoroacetate required to kill different animals varies from 0·06 milligramme per kilogram of body weight in the dog to 500 milligrammes per kilogram of body weight in the toad. The rabbit requires 0·2 milligramme per kilogram and the rat requires five milligrammes per kilogram approximately. The dose required to kill a man is probably about five milligrammes per kilogram of body weight.

The first signs and symptoms of fluoroacetate poisoning which usually appear after an interval are vague feelings of discomfort and scattered tingling sensations with areas of numbness. These are usually accompanied by nausea and vomiting and are succeeded by spasmodic contractions and by the twitching of voluntary muscles. Later, violent convulsions at frequent intervals develop. Death may occur suddenly, as a result of cardiac failure due to ventricular fibrillation, or it may be the result of long and repeated convulsive seizures, resulting in exhaustion and respiratory failure. In rabbits, death appears to be due to the rapid onset of ventricular fibrillation after a violent convulsion. In dogs, death is typically the result of repeated and prolonged convulsions on the respiratory system. The heart and lungs of rabbits poisoned with 1080 have a characteristic post-mortem appearance. The lungs are a bright red colour. The heart is a darker red and the surface blood vessels are dilated and stand out from the heart surface.

Sodium fluoroacetate is also a very toxic systemic insecticide. It has been effectively used against the larvæ of the citrus gall wasp *Eurytoma feelis* (Gir.).

Conclusion.

It is obvious that future research will produce more effective pesticides. It should be the duty of the various governing authorities to see that the toxic effects of these new pesticides are thoroughly understood before they are released. If necessary, their use by the public should be either controlled or supervised. It should also be the aim of the research chemists to produce effective pesticides that have a low toxicity to human beings.

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SO-CALLED "ACIDOSIS ATTACKS" IN CHILDREN.[1]

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Brisbane.

UNDER the title of "acidosis attacks", it is proposed to discuss the medical histories of a group of children examined in one practice in a single year, each of whom exhibited at intervals such symptoms as nausea, vomiting, fever, headache and abdominal pain. Although in no two cases were the clinical pictures exactly alike, yet they all had enough in common to justify one in regarding the patients as suffering from a single disease, or at least as exhibiting the same sort of bodily reactions.

As an example we may take Kevin's case. This little lad, who was nine years old when he first came, had started about eighteen months earlier to have severe vomiting bouts, during which—in his mother's words—he "kept nothing down", and complained of fullness in the stomach and frontal headaches. The attacks usually began in the mornings, the general pattern being that he woke with headache and after a while became nauseated and vomited. For the next six or eight hours he would lie in bed pale and miserable until the attack passed off. For a few weeks thereafter until the next episode he would be in perfect health, displaying what his mother described as "a terrible lot of energy", being quite tireless, eating and sleeping well. An only child, he was of nervous disposition and never still. His mother herself had suffered similar attacks as a child and had subsequently developed migraine. Two maternal aunts and one uncle were affected in like manner.

Nomenclature.

Two generations ago Kevin's trouble would have been described as "bilious attacks" and a little later as attacks of "acidosis". The expression is now going out of fashion, and very properly, because it is incorrect. It was first used by Naunyn in 1906 to describe an accumulation of ketones in the body as indicated by their presence in the urine. It so happens that in the case of diabetes, about which he was writing, the presence of ketones is associated with an excess of acid; but the impression soon became general that the presence of ketones always signified acidosis. This is actually not so, and such a view has been untenable for many years. Ketones are normal products of fat metabolism produced almost entirely in the liver and oxidized in the muscles. Over-production of ketones occurs when the liver glycogen content falls below a critical level—that is when, owing to shortage of carbohydrate reserves, excess fat has to be oxidized to supply the body's energy needs. However, the tissues cannot deal with ketones, nor can the kidneys excrete them, beyond a certain rate. Increasing keto acids then compete for base in the blood-stream and may be a factor in the development of acidosis; but only one factor. "Ketosis", which may be regarded as evidence of a diminished glycogen content of the liver, has not necessarily anything to do with "acidosis", which signifies a diminution in the alkali reserve of the body. In fact, when it is accompanied by excess vomiting of gastric hydrochloride acid, it is possible, though rare, for ketosis to occur with systemic alkalosis.

However, there is really nothing to be gained by dropping the term ""acidosis" and substitutuing "ketosis", to describe the recurrent vomiting attacks, for ketonuria is very common in childhood and a frequent clinical finding in starvation and fever.

Physiological Changes.

There is some uncertainty about the nature of the physiological alteration that forms the background for the attacks. Vomiting of cerebral origin may be the

[1] Read at a meeting of the Section of Pædiatrics, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August 20 to 27, 1955.

primary fault, with ketosis inevitably following. Yet it has been observed that ketosis appears much earlier in the cyclical attacks than it does, say, in the vomiting of high intestinal obstruction. Also, on some occasions the child will be found ill with pallor, nausea, abdominal pain and acetone in the urine, even though vomiting has never occurred.

Some have held that accumulation of keto acids in the serum, with a resultant fall in bicarbonate levels, is the initial change, and that vomiting is a secondary or compensatory mechanism to lose the acid radical chloride —that is to say, that ketosis causes the vomiting and not *vice versa*. Yet this also cannot be the whole truth, in the light of Professor Alan Moncrieff's observations on epileptic children being treated with ketogenic diets. He noted no single instance in which, with manifest ketonuria, attacks of vomiting had occurred, although transient nausea had been recorded.

Other writers have noted that when the attacks follow an emotional disturbance, there may be a lapse of about forty-eight hours between the emotional shock and the onset of symptoms. It may well be that certain factors entail a sudden call on liver glycogen, reducing it below the critical level so that excess fat catabolism and ketosis appear. These factors could conceivably be mild infections or emotional upsets, although the regularity and spacing of the episodes in many children suggest a rhythmic metabolic change.

In 1951 Payne investigated sugar metabolism in children with cyclical vomiting and found that although the glucose tolerance curves were normal the response to adrenaline was unusually high. He took this to indicate an increased ease of glycogen mobilization. He also found that at the beginning of an attack the blood sugar level tended to be high; later it was low, perhaps from starvation. Payne advanced the theory that the first step was the excessive utilization of blood sugar by nervous, highly-strung children. This led to depletion of liver glycogen and then to hypoglycæmia. This in turn called forth excess adrenaline secretion, leading to further depletion of liver glycogen, further fall in blood sugar level and further adrenaline production. He attributed all the symptoms to excess adrenaline.

It seems doubtful whether this explanation either is the correct one, and it can honestly be said that the essential nature of these attacks is still a mystery.

Clinical Picture.

Perhaps the best way to convey the variability and yet the essential similarity of the condition as revealed by different children is to relate very briefly a few of the clinical histories.

For example, Graham provides an interesting study of the type of child who suffers from this disorder. Now eight years old, he has for the past five years endured episodes of headache and pain in the abdomen occurring at irregular intervals of one to six weeks. Until two years ago he used to vomit clear fluid during the attacks, but this has now ceased. His trouble begins rather constantly about 4 a.m. and is accompanied by a slight rise in temperature; but in spite of this he insists on going to school at 8.30, looking tired and pale and having eaten no breakfast. The elder of two adopted children, Graham's family history is unknown, but he has had no previous illnesses and can tolerate fats normally. He is at all times an excitable, self-conscious child who worries badly. His foster-mother described him privately as "a fussy perfectionist, impossible to live with". Graham was seven pounds below average weight. Careful examination, including a visit to an ophthalmologist, revealed no evident cause for his headaches.

Lawrence, aged seven years, was brought to Brisbane from a country town with the history that over the last eighteen months he had sustained attacks fairly regularly every few weeks. On these occasions he would become limp and pale, usually complain of headache and generally vomit. Sometimes he was feverish. He did not want to eat, but would, rather curiously, drink a little milk while the attack was on. He chiefly wished to be left alone, and when he was very severely affected, even the vibration of anyone walking over the floor would upset him. Recovery was spontaneous in two days. Lawrence was five pounds below average weight and somewhat anæmic. His father was said to be delicate and to suffer from very severe headaches.

Some children are fortunate enough to have very brief attacks.

Robin, aged eight years, had suffered over the past twelve months from what were described as "bad headaches and bilious turns", recurring about once a month and becoming worse. His trouble tended to start in the late afternoon with headache, followed by vomiting; but he would become drowsy during the evening, sleep heavily overnight, and wake feeling well. He was said to have a "tremendous amount of nervous energy", and would work and play very hard, but "flop" afterwards. His mother commented that he was not the happy child he used to be, and thought he had lost weight. He ate a lot of fruit, but had not much appetite for anything else. His paternal grandfather had always suffered from severe headaches, and Robin's father likewise, but at long intervals. Robin proved to be of average weight for his age, and he looked well; but he was nervous and emotionally unstable and laughed too easily.

Sometimes fever assumes prominence as a symptom.

Robert, who was nine years old, complained of a series of episodes of fever, vomiting and abdominal pain occurring frequently enough to interfere with his schooling. The attacks had raised the suspicion of appendicitis until the most recent occasion, when he had a temperature of 100·2° F. and vomited for some days, but had no pain at all. His mother was subject to severe headaches. Robert was a thin lad, 11 pounds below average weight. Thorough examination and a variety of investigations revealed no abnormalities. He was examined again a fortnight later after another attack of fever and headache and "sickness in the stomach" lasting a few days, but again without abdominal pain. Once again investigation gave negative results.

Vomiting is not an essential symptom, as the following case history suggests.

Carla, a pleasant little girl, aged nine years, had suffered in the past six months four attacks of abdominal pain, headache and collapse. They always began in the early morning before rising. She woke looking pale and languid, and severe frontal headache usually preceded the abdominal pain, which was generally epigastric. She had no appetite at all for twelve hours or so, then started to eat and picked up rapidly. In the latest attack, but not in the previous ones, she had vomited four times. Her father suffered from migraine.

You have listened patiently to six clinical histories, enough perhaps to give you a sort of composite picture of the condition under discussion. Admittedly the symptoms have been in each case relatively mild and in no instance bad enough to bring the sufferer into hospital. Yet it would surely be a mistake to restrict the term "cyclical vomiting" only to the alarming upsets accompanied by profound ketosis and dehydration. Attacks come in all grades of severity, and vary so much in a single child from time to time that our concept of the disorder must also include the sort of mild recurring episodes that have just been described.

Diagnosis.

In the absence of physical signs between attacks, diagnosis becomes a matter of a carefully taken history. One distinguishing feature is the regularity of the attacks, which often occur about the same number of days, weeks or months apart and in any particular child have a habit of starting at the same time of the day. There also tends to be a general resemblance between one attack and the next, though they may differ in severity. Finally, the rapid termination of the episodes and the good general health and abundant energy between times are most suggestive.

There is often a particular family background. Franklin writes as follows:

> Many of these themes recur in our talks with parents: the over-valued child; the child born to take an already established place in the family business or profession, old before its time; the child who must not suffer as its parents have done, and who is therefore denied the influences that have formed their characters; the child who is used as a pawn in the chess game between two sides of a family; the child who is too good to mix with the neighbouring children; the child who

was not wanted, the knowledge of whose conception was a cause of panic and dismay, and who has survived such a battering by abortifacients that his parents can hardly look him in the face for guilt. And then there are the whole group of broken homes that lack security, some broken by death, some by separation or divorce.

It goes without saying that recurring episodes of organic disease must be excluded before one can make a diagnosis, and this is hardly possible without seeing the child during an attack. Common conditions capable of producing a similar pattern of headaches, fever, vomiting and abdominal pain are recurring bouts of acute tonsillitis, of appendicitis and of pyelonephritis—readily identified by their special physical signs.

When the history is relatively short and the vomiting occurs particularly in the mornings, the possibility of intracranial neoplasm must be kept in mind. However, the tendency of the child with cyclical vomiting to be bright and well between attacks is in sharp contrast to the ways of a child with a cerebral tumour, whose condition changes little from day to day.

Diagnosis becomes more difficult when one faces—perhaps in a hospital bed—a child in a really severe attack of cyclical vomiting with ketosis and dehydration. Possibly the history is scrappy, the parents are absent and the child is too ill to care. Appearances may suggest the early stages of overwhelming gastro-enteritis, accidental poisoning, diabetic coma or high intestinal obstruction. The last possibility may cause so much uncertainty that in spite of the flat, empty abdomen, laparotomy is seriously considered and even undertaken.

Ætiology.

For the proper management of this disorder it is necessary to be as clear as possible about its essential nature. It has been held, for example, that the repeated attacks are due to recurring infection, probably because of the rise in temperature that is sometimes a feature. However, such claims fail on the absence of fever in so many attacks, the absence of leucocytosis, the absence of any focus of infection, the regularity of the attacks in some children and the short duration, sometimes only a matter of hours, in others.

It has also been held that allergy plays a part in the causation of the attacks, but only, I suspect, by those who ascribe to allergy any unexplained medical phenomenon. Hamilton, referring to claims of food allergy, states that alterations in diet and skin tests do not seem to support this view. Indeed, it is the common experience of parents that their child will often have his attacks for no apparent reason and certainly without any change in the usual pattern of his diet.

Faced with the suggestion that the attacks are due to emotional stress, we are on more difficult ground, for it is essentially true to say that cyclical vomiting does occur in children of a particular type. They have been described as "thin, hypotonic subjects, rather above average intelligence, energetic, but tiring easily and taking things rather hard as a whole". In such children emotional stress may, and I am sure often does, set off particular episodes; but it can hardly be blamed for all attacks, many of which seem to arise for no special reason at all.

For example, Percy, a boy aged six years, has suffered bouts of vomiting almost since infancy. They now appear at intervals of six to eight weeks. He is always thin, active and energetic, but even between times he does not sleep very well and tires easily. His attacks usually begin in the early morning, when he wakes looking pale and not wanting breakfast. He may vomit, and either drags himself to school or stays listlessly at home. He usually vomits again in the evening after being forced to eat dinner, and may go on like this for a week or even more. His attacks have been worse and more prolonged since he has come to live in a larger town and has been attending a new school which he dislikes. Many of the attacks now seem to be provoked by emotional upset. His father's sister has migraine, and his father suffers from "bad heads", while his mother used to have bilious attacks as a child. Percy is a thin, nervous, sensitive child, five pounds below average weight, but otherwise he appears healthy.

The ætiological significance of fat intolerance is interesting. Sheldon, in the latest edition of his textbook, ascribes cyclical vomiting to "the recurrent breakdown of fat metabolism" and condemns the habit of feeding the child with cream or emulsions as "laying the foundation for the next attack"; yet on the next page he refers to the experiments, already mentioned, in which children known to be subject to these attacks were intentionally fed on ketogenic diets without the provocation of symptoms. On the other hand, it is undoubtedly true that many of the children who suffer from periodic vomiting dislike fats and are upset by them, and some of them suffered from fat intolerance in infancy. Perhaps it would be correct to say that fat intolerance relates rather to the type of child under consideration than to the actual mechanism of the attacks.

Consider the case of Jonathan, now five years old. According to his mother he has "always had liver trouble"—that is, he cannot tolerate much fat, anything more than a few ounces of milk or a scrape of butter upsetting him. Every month or so he has an episode lasting as long as a week during which he is listless, complains of abdominal pain and occasionally vomits. Sometimes the pain is enough to wake him at night. Even when well he is a quiet child, but has a fair appetite. He vomited a lot as a baby and was highly nervous. He quite likes school now, but it was a terrifying business getting him started.

However, the most significant question in this discussion is the relationship of cyclical vomiting to migraine. Let us refer again to Jonathan with the fat intolerance.

He is nine pounds below average weight, and in build and temperament resembles his father, about whom his wife said: "He is a quiet, gentle man who immediately feels sick in the stomach if there is any trouble in the family." Both Jonathan's father and most of the father's family suffer from migraine.

Consider also the two following cases, the last of the series.

Gary, aged five years, had for years been subject to violent bilious attacks every few months. In the last three attacks, but not in any of the earlier ones, there had also been temporary visual disturbance lasting perhaps half an hour. During this time his eyes would go glassy and his head and eyes would turn sideways, but without nystagmus. He could speak and answer questions, but for a short period complained of not being able to see properly and of headache over the right eye. During the attack he was noticeably pale. The vomiting, though severe, never lasted longer than one day, and often less.

Finally Elizabeth, who was ten years old, had been suffering from periodic severe headaches for months when she was first taken to the doctor. She was a clever child, who topped her class of 70 at the State school and seemed to fill all her day with useful activities. She read a great deal, learned and practised music assiduously, spent Sunday polishing the family silver and (I suspect) played very little. Her mother regarded her as "definitely livery", because of her sallow skin, and some years earlier she had been treated on a fat-free diet by her family doctor, who had attributed to an "over-loaded liver" the bilious attacks she then suffered. Her mother had migraine and "suffered a lot with her head".

On talking to the parents of these children, one is struck by the frequent history of migraine or at least of severe recurring headaches in near relations, who often themselves suffered severe bilious attacks in childhood. The association has drawn from various writers such comments as Hamilton's, who wrote: "The relationship between cyclical vomiting and migraine is universally recognized; indeed the one frequently merges imperceptibly into the other." Similarly, Milton Senn wrote of migraine as follows: "In young children the headache may be a minor feature, but nausea and vomiting may be marked. Migraine may later replace attacks of recurrent vomiting."

Indeed, recognition of the link with migraine goes back to Samuel Gee, who in 1882 described the first nine cases of cyclical vomiting and included this feature in his description. Franklin also, describing "periodic disorders of children", remarks on the way the symptom pattern alters as the years pass, the best-known change being the gradual development of migraine by the recurrent vomiter.

May it then be suggested for your consideration that what have variously been described in the past as bilious attacks, acidosis, ketosis, cyclical vomiting and periodic disease are, in fact, the juvenile form of migraine, hitherto usually regarded as an adult disease?

Treatment.

Something can be done to relieve the misery of the attacks. If they are at all severe, rest in bed is wise. Whether toys, games, visitors or wireless are permitted depends on the wishes of the child. Usually he prefers to lie alone in the dark and keep quiet. He will not need food, but he does need what fluids he can tolerate, generally preferring soft drinks. Milk is quite wrong. Glucose water, beloved of hospitals, is slightly nauseating by mouth, even to the healthy, but may be required in the more severe cases by the subcutaneous drip method of administration.

Sedation helps these children. Until recently phenobarbital was the drug of choice, administered in quarter-grain or half-grain doses every four hours. Once properly sedated, they sleep heavily and wake with the worst of their troubles over. Recent experience suggests that chlorpromazine, one milligramme per kilogram of body weight every six hours, may be even better than phenobarbital, for relieving both the vomiting and the associated symptoms.

However, the doctor has responsibilities beyond the mere management of the attacks. He has to inculcate, both in parent and in child, a healthy, tolerant, matter-of-fact attitude of mind towards the whole condition. Success in this demands many qualities which we can do little more than mention here. It requires, as Franklin said, "the firm touch of the confident physician, certain of his diagnosis". It requires that the parents, and the child too if he is old enough, be told the truth about the disorder, its recurring nature, its nuisance value, its freedom from real danger. They have to learn not to fear it but to accept it, and to avoid wandering from doctor to doctor in pathetic, fruitless search for a cure.

Just as parental anxiety over the child's health can set up emotional tensions in the home and thus establish a vicious circle, so can a calm, courageous attitude give him the necessary confidence. If in addition he can be promised relief from unpleasant symptoms, he may be able to come to regard his troubles as little more than "emotional growing pains" which doctor and parents can help him to endure. For those who are under continual stress and whose attacks are coming frequently in consequence, phenobarbital over a long period in doses of 0·5 milligramme two or three times a day, or chlorpromazine in two daily doses each of 0·5 milligramme per kilogram, is likely to be beneficial.

Conclusion.

The case histories of ten children have been presented. I want to leave you with the impression that they have enough in common to justify the use of a single title which could well be "juvenile migraine". I also want you to feel that, though he understands little of the nature of the disorder, the pædiatrician can still play a substantial and useful role in its recognition and management.

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COXSACKIE INFECTION IN QUEENSLAND.

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SINCE their discovery in 1948, viruses of the Coxsackie group have been found in many countries, and there has been much discussion of their relation to human illness. Interest in Queensland was stimulated by the occurrence of several outbreaks of illness in which a Coxsackie ætiology was suspected, and the study was extended to include many sporadic cases also. Altogether 125 patients were investigated. From the fæces of 79, seven strains of Coxsackie virus were isolated, and the serum of 63 was examined for neutralizing antibodies.

Materials and Methods.

Observations were made on the 97 North Queensland patients, and specimens were collected from them by the staff of the Innisfail Field Station of the Queensland Institute of Medical Research. Material in which a virus was sought was maintained on dry ice during transfer to Brisbane and storage until tested. For the 28 Brisbane patients we enjoyed the cooperation of a number of physicians.

Fæces were prepared by a concentration method, similar to that described by Stanley and Hayes (1950). Before a "Spinco" ultracentrifuge became available in Brisbane in mid-1952, the unconcentrated supernate of the fæcal emulsion, after treatment with streptomycin and penicillin, was used for inoculation. Blood samples were ground with alundum, and diluted if necessary with physiological saline.

The "Q.I.M.R." strain of mice was established from stock obtained in 1949 from the Walter and Eliza Hall Institute, Melbourne. Most of those used were less than twenty-four hours old; they were inoculated intraperitoneally. Blind passages were rarely made if there were no signs of infection in the mice primarily inoculated. For passaging a virus, the tissues selected in the earlier work were muscle and brain; but more recently, skinned, beheaded and eviscerated carcasses were used. Often carcasses were stored at −15° C. until passaged. Streptomycin and penicillin were added to inocula to concentrations of 0·5 milligramme and 100 units per millilitre.

Isolated strains were typed and neutralization tests carried out in the Institute of Epidemiology and Preventive Medicine, Sydney. For the neutralization tests, a standard virus suspension was mixed with varying serum dilutions as described by Stanley *et alii* (1951). The mice used for these tests were the Prince Henry mice described recently by Stanley *et alii* (1954).

Convalescent serum was usually screened at a dilution of 1/80. If no neutralizing antibody was found, the result was regarded as negative and no further testing was done. If the result was positive at 1/80, then both acute and convalescent sera were titrated. Titres were calculated by the method of Reed and Muench (1938). Pooled serum and some individual sera were tested at 1/20.

Brisbane Cases.

Isolation of a Group A Virus.

Between October, 1950, and May, 1954, the fæces of 19 Brisbane patients were investigated for the presence of Coxsackie virus. The final diagnoses in these cases were as follows: aseptic meningitis, seven; encephalitis, four; Bornholm disease, four; poliomyelitis, three; mumps, one.

In one case of aseptic meningitis, a Coxsackie virus of group A, type 7 was isolated:

CASE I.—H124, a girl, aged seventeen years, assisted in the care of children at a Red Cross Home at Redcliffe, near Brisbane. She developed backache on the evening of October 24, 1950, which became worse over the next two days. On October 27 she was ill, with a temperature of 101·7° F. and a pulse rate of 120 per minute, and was admitted to the Brisbane Base Hospital under the care of Dr. H. R. Love. She had slight neck stiffness for a time, but no muscle weakness. The cerebro-spinal fluid was clear at a pressure of 100 millimetres of water. It contained 40 cells per cubic millimetre, of which 90% were polymorphonuclear cells and 10% mononuclear cells, and 50 milligrammes of protein per 100 millilitres. The temperature rose to 102·6° F. on October 28, but became normal next day. General improvement was rapid, and she was discharged from hospital on November 3. The fæces from which the virus was isolated were collected on November 2. Neutralizing antibodies to her own virus were found in the serum of H124 to titres of 1/540 (eleventh day[1]), 1/380 (thirty-second day) and 1/95 (297th day). It is, however, not possible to determine whether the patient's infection with the Coxsackie virus was responsible for the illness or merely coincidental. (The serum on the thirty-second day neutralized also a B2 Coxsackie virus to a titre of 1/160.)

H124 was one of many patients who developed aseptic meningitis in Brisbane in the latter half of 1950. Parry (1951) has described 11 at the Brisbane Children's Hospital in August and September, and referred briefly to 17 subsequent cases; in none of these were specific virological studies made. Parry's patients came from scattered locations, mostly on the outskirts of Brisbane, including Redcliffe. Parry considered that the cases were too numerous to have been non-paralytic poliomyelitis, for at that time paralytic poliomyelitis was very infrequent in Brisbane. But the diagnosis which she favoured—lymphocytic choriomeningitis—seems improbable because of the mild nature of the illness, the wide geographical scattering of the cases and the absence of any special mouse contact. It is tempting to regard the outbreak as due to Coxsackie infection; the distribution mainly in the unsewered outer suburbs of Brisbane would accord with an intestinal agent. However, the isolation of a group A Coxsackie virus from one of the three patients investigated at that time is very slender evidence to offer, particularly as the relation of group A viruses to human disease, apart from herpangina, has not been clearly established.

During the period of H124's illness in Brisbane, the same type of Coxsackie virus was isolated from the fæces of two patients in Sydney (Stanley *et alii*, 1951, 1953*a*). One suffered from Bornholm disease with meningitis, the other from poliomyelitis.

No virus was obtained from cerebro-spinal fluid (four subjects), blood (four subjects) or brain (one subject) in various Brisbane cases of infection of the central nervous system.

- Investigation of Serum for Group B Antibodies.

The results of tests for neutralizing antibodies against group B Coxsackie viruses in the serum of 12 Brisbane patients and in two serum pools are recorded in Table I.

The patients included four with encephalitis, two with aseptic meningitis, and one diagnosed as suffering from Bornholm disease. (The fæces of three of these patients were tested; only from H124, mentioned above, was a virus obtained.) Four other patients, who had been admitted to hospital with the diagnosis of influenza in October, 1953, during an epidemic, were tested on the suspicion (which was not borne out) that Coxsackie viruses might be contributing to the epidemic; in three of the four cases the diagnosis of influenza was confirmed by specific tests. Two pools of serum of healthy persons were obtained through the kindness of Dr. A. E. F. Shaw, Director of the Red Cross Blood Transfusion Service, Brisbane.

In no case was there demonstrated a rise in antibody titre indicative of recent infection. However, antibody to group B, type 2, was present in four of seven subjects tested, to type 4 in three of nine subjects, and to both these types in the two serum pools. Antibody to type 3 was found in only one of seven individuals, and not in the pools. Antibody to type 1 could not be demonstrated in either individual or pooled serum. If one may judge from these small totals, it seems likely that, in Brisbane, Coxsackie viruses of group B, types 2 and 4, have wide distribution compared with types 1 and 3. The Coxsackie antibody levels in the pooled human serum collected in Sydney and Brisbane are almost identical; but whilst group B, type 3 antibody was not detected in the Brisbane pools, it was consistently demonstrated in Sydney pooled human serum after the outbreak of "Sydney encephalitis" in 1951-1952 (Stanley *et alii*, 1953*b*).

As a Coxsackie virus antigenically related to group B, type 3, was responsible for this outbreak in Sydney, the Brisbane case of encephalitis in which antibody to this type was demonstrated is given in detail.

[1] In this paper the day of onset is regarded as the first day.

TABLE I.

Coxsackie Neutralization Tests on Brisbane Serum.

Subject.	Sex of Patient.	Age. (Years.)	Date of Onset.	Day from Onset when Serum Collected.	Reciprocal Titres.				Diagnosis.
					B1.	B2.	B3.	B4.	
H124	F.	17	24.10.50	32		160			Aseptic meningitis; Coxsackie A7 virus isolated.
L937	F.	22	29. 4.51	81	<20	<20	<20	<20	Encephalitis.
M938	M.	33	15. 6.51	35	<20	<20	<20	<20	Encephalitis.
G939	F.	68	4. 7.51	15	<20	<20	<20	<20	Hypertensive encephalopathy.
R942	F.	18	30. 9.51	5				160	Aseptic meningitis.
				9		190			
				30	<80	230	<80	105	
J207	M.	47	4. 2.52	4			40		Bornholm disease.
				31			40		
R417	F.	1⁷/₁₂	19. 4.53	6			235		Encephalitis.
				23			550		
S686	M.	11	30. 9.53	17	<20	140	<20	<20	Fatal encephalitis, probably post influenzal.
T638	M.	19	30. 9.53	3				640	Final diagnosis, influenza B.
				23				>1280	
F637	M.	19	1.10.53	2				320	Influenza B virus isolated from throat.
				22				600	
N636	M.	18	1.10.53	22				20	Final diagnosis, influenza B.
E639	M.	18	1.10.53	22		140		>20	
Pool of 30 30.6.53 to 1.7.53					<20	>20	<20	>20	
Pool of 18 17.11.53					<20	>20	<20	>20	

CASE II.—R417, a female infant, aged nineteen months, who lived at Redcliffe, became feverish on April 19, 1953, and a few hours later had a convulsive seizure. She was admitted to the Brisbane Children's Hospital under the care of Dr. F. Arden. The convulsions continued after her admission until they were controlled by phenobarbitone given intramuscularly and potassium bromide and chloral given by mouth. The temperature was remittent, reaching 102° F. each afternoon until and including April 22. She was cutting three teeth, and there was some inflammation of the tonsils and suspicion of *otitis media*. Stiffness of neck and back were noted on some occasions, but were absent on others. "Seclopen", 300,000 units, was administered daily.

On April 22 convulsions recurred, lasting forty minutes and involving the left arm and the left side of the face. These were repeated frequently during the day. Phenobarbitone was freely administered by the intramuscular route. Afterwards the left arm and leg were flaccid, and left facial paresis was present. Twitchings were noted in the left arm and left side of the face. Lumbar puncture yielded normal cerebro-spinal fluid containing no cells. Next morning the temperature was normal. There was gradual improvement, but a left-sided paresis remained. At the time of her discharge from hospital on May 16, movement of the left leg had returned, but there was still paresis of the left arm. The fæces were not tested.

The presence of high-titre antibody to group B, type 3 virus suggests that this virus may have caused the illness, but this cannot be established, particularly as no serum antedating the formation of antibody was examined—the first specimen was taken on the sixth day of illness. Dr. Arden informed us that R417 was one of six young children with encephalitis whom he examined within a month. These cases invite comparison with the Sydney series of Stanley *et alii*. However, the Brisbane cases occurred at a much earlier age and their severity was much greater; four of the patients died, and the other two were left with the sequelæ of hemiparesis or epileptic attacks.

North Queensland Cases.

1952.

A number of cases, diagnosed clinically as Bornholm disease, occurred in Innisfail and neighbouring localities in January and February, 1952. Fæces from five patients and throat washings from two were inoculated into infant mice. A Coxsackie virus was isolated from the fæces of one patient, M222, and proved to belong to group B, type 3.

CASE III.—M222, a tailoress, aged eighteen years, lived near Innisfail, but had been on holiday in Brisbane and other centres in southern Queensland, returning home on January 19, 1952. On January 21 she developed a headache which aspirin would not clear up. Next day she went to work as usual, but by lunch time had mild diarrhœa, sore throat, and a temperature of 102° F., and was admitted to hospital under the care of Dr. W. N. Markwell. There, epigastric pain which radiated to her chest and caused difficulty in breathing came on. She complained that the pain "seemed to be inside her chest wall". Her headache became much worse, her eyes sore and her vision affected. Movement of her right leg gave her pain.

On examination of the patient her chest was clear, and no nuchal rigidity, no weakness in the legs or other muscle groups, no adenitis and no significant eye signs were detected. Her reflexes were normal. She was treated conservatively, and after five days in hospital was discharged, cured.

Paired sera from M222 showed a notable rise in the titre of neutralizing antibody to her own strain of virus (Table II). The convalescent sera of two other Innisfail patients who became ill in January, 1952, did not contain antibody to this strain.

It is noteworthy that the infection of M222 occurred in the same period as the encephalitis epidemic in Sydney, already mentioned, and that it was due to a similar virus type.

The following infection probably arose in the laboratory:

CASE IV.—A research officer (R208), who had been passaging the M222 virus in mice, became ill on March 16, 1952, with shivers and generalized pains and aches. However, she worked on March 17 and for part of the next two days. She had pain in the eyes, soreness in the muscles of the neck and pallor. On the evening of March 19 the temperature rose to 101° F. On March 20 the pains had mostly disappeared, leaving a feeling of exhaustion. She returned to work, quite well, on March 24. Examination of early convalescent serum revealed an increase in neutralizing antibody against the M222 virus when compared with serum taken a month before the illness (Table II).

Specific antibody was still abundantly present in the serum of M222 and R208 three years after their illness (Table II).

1953.

Doherty (1956) has reviewed a series of febrile cases which he observed in the area served by the Innisfail Field Station from September to December, 1953. A suspicion that some were Coxsackie infections led to the submission of specimens of fæces and blood to Brisbane and paired sera to Sydney. Coxsackie viruses of group B, type 4, were isolated from the fæces of four patients out of 12 tested. Clinical records of these four are included in Doherty's paper. No virus was isolated from the blood of nine patients, although it was present in the fæces of two of them.

Serum from 30 patients was examined for neutralizing antibody against one of the isolated North Queensland strains (G703). The results are recorded in Table III, and attention may be drawn to certain points:

1. In two of the patients from whose fæces a virus was isolated, G703 and H700, the illness was associated with a striking rise in specific antibody titre. In a third, J699, there was a smaller, but definite, rise.

2. In B697 no antibody was detected in acute or convalescent serum, although virus was recovered from fæces.

3. None of the other 26 patients tested serologically showed an antibody rise comparable with that of G703 and H700 (and M222 of Table II). However, in two of them, C698 and G704, there was a fourfold rise which might be considered indicative of recent infection.

4. In eight cases, which were distributed from Gordonvale to Tully, antibody was present to a titre of at least 1/80 in the serum collected early in the illness. Therefore, infection with a group B, type 4 Coxsackie virus must previously have been widespread in the district.

5. In three cases, antibody was demonstrated in convalescent serum, no early serum being available.

6. In H700, antibody persisted for at least sixteen months after the illness.

The serum of 25 patients was tested also for neutralizing antibody against a group B, type 2 virus. In no case was a significant rise found, but in four cases the presence of antibody early in the illness showed that this type also had previously been in the district.

Investigation of Pooled Serum, 1951–1953.

Ten pools were prepared from the serum of North Queensland patients who had been under investigation for "fever". Each pool consisted of serum from 15 to 99 individuals, and covered a period of three months, the periods extending from July-September, 1951, to October-December, 1953. Tests for neutralizing antibodies to Coxsackie virus group B, types 1, 2, 3 and 4, were carried out at a final serum dilution of 1/20. The results (Table IV) confirm the observation made with individual sera that group B, types 2 and 4, have wide distribution in this area. Type 1 antibody could not be demonstrated, and type 3 appeared only in small amounts compared with types 2 and 4.

1954.

Isolation of Virus.—During 1954, fæces from 43 patients from the Innisfail district were examined for the presence of Coxsackie virus. These patients suffered from "fever", the cause of which was not apparent at the time. An occasional patient had neck stiffness, but none had pleurodynia. A virus of a type not yet determined was isolated from one patient, W864, a female, aged six years, who

TABLE II.

Investigations for Coxsackie Infection, North Queensland, 1952.

Patient.	Sex.	Age. (Years.)	Onset.	Tests for Virus in Fæces.		B3[1] Neutralizing Antibody in Serum.		Comment.
				Day Collected.	Result.	Day Collected.	Reciprocal Titre.	
P940	F.	14	5.1.52	4	−	29	<20	
B941	F.	35	8.1.52	8	−	30	30	
M222	F.	18	21.1.52	4	+	3	<20	
						100	450	
						1102	530	
R208	F.	23	16.3.52			0	<20	Probable laboratory infection.
						9	170	
						1032	525	

[1] The M222 strain of virus was used in the tests.

became ill on May 15, 1954, and whose history is recorded by Doherty. Neutralizing antibody to her own strain was not found in her convalescent serum, taken on the fortieth day.

Serological Observations on Townsville Serum.—In February, 1954, an epidemic of dengue prevailed in Townsville. While the nature of the epidemic was still in question, paired sera from 16 patients, obtained through the courtesy of Dr. R. A. Rimington, were tested for antibody to Coxsackie virus group B, type 4. In no case was there a significant rise; but the presence of a titre greater than 1/80 in the "acute" serum of five patients indicated that infection with this Coxsackie type had previously occurred in Townsville, as in the Innisfail district. Two subjects were tested also for group B, type 2 antibody. One was found to have titres of 1/160 and 1/490; the other had a convalescent titre of less than 1/80.

Distribution of Cases by Sex, Age and Season.

Virus was isolated from the fæces of three of 54 males examined and from four of 25 females. The difference is not significant.

The age distribution is recorded in Table V. Although the proportion of successful isolations was greater in the first two decades of life (five out of 35) than in later years (two out of 44), the difference is not significant.

During the warmer months, from October to March, virus was isolated from six of 45 patients, during the colder months from one of 34. The difference is almost significant, being 1·8 times the standard error.

Behaviour of Viruses in Mice.

H124 Strain—Group A, Type 7.

Infant mice were inoculated in Brisbane with the fæces of H124, but no virus was obtained. In June, 1951, the

TABLE III.

Results of Neutralization Tests on North Queensland Serum against Coxsackie Virus Types B2 and B4.

Patient.	Sex.	Age. (Years.)	Date of Onset.	Day when Serum was Collected.	Reciprocal Titres.		Comments.
					B2. Strain T80.	B4. Strain G703.	
D646	F.	38	14. 9.53	4		130	Final diagnosis influenza B.
				46		400	
V648	F.	48	15. 9.53	3	184	69	
				45	80	106	
S754	M.	32	6.10.53	29	<80	142	
				56		68	
C649	M.	17	9.10.53	4	160	400	Final diagnosis influenza B.
				41	216	260	
S795	F.	12	26.10.53	2		240	
				29		500	
S732	M.	9	29.10.53	1		530	
				51	<80	720	
P749	M.	35	31.10.53	10		450	
				62	80	970	
C739	M.	24	10.11.53	3		120	
				36		50	
H701	M.	36	11.11.53	2		113	Final diagnosis probably influenza B.
				56	<80	160	
L706	M.	22	12.11.53	2	>1280	52	
				26	870	40	
E740	M.	20	14.11.53	10		179	
				52	<80	40	
G703	M.	37	16.11.53	3		<20	B4 Coxsackie virus isolated from fæces on fourth day of illness.
				54	<80	765	
C698	M.	25	16.11.53	1		40	
				33	>80	160	
M705	M.	21	17.11.53	3		106	
				43	50	60	
J699	F.	34	17.11.53	4	40	<20	B4 Coxsackie virus isolated from fæces on seventh day.
				36	105	132	
				469		60	
B697	M.	12	21.11.53	5		<20	B4 Coxsackie virus isolated from fæces on fourth day.
				39	<80	<20	
G704	M.	12	21.11.53	5		<20	
				39	<80	80	
H700	M.	6	22.11.53	1		<20	B4 Coxsackie virus isolated from fæces on third day.
				51	<80	320	
				475		1280	
M750	F.	47	2.12.53	6	132		
				39	134	<80	
M736	M.	20	6.12.53	5		130	
				37	<80	120	

fæcal sample, which had been stored for seven months at −20° C., was sent to Sydney. The isolation of virus from this material by the concentration method has been reported by Stanley and Dorman (1953).

M222 Strain—Group B, Type 3.

The M222 strain easily became adapted to infant mice, the mortality rate being high in the second and third passages. Affected infant mice showed cessation of growth, general weakness, paralysis (most pronounced in the hind limbs), tremor, head retraction, muscle spasms and semi-coma. In some mice that died after fourteen days, or recovered and were killed later for examination, numerous small white patches were seen in the brain. On microscopic examination, the most evident changes were encephalitis and necrosis of fat. Occasionally mild, focal myositis was found. Further information on the M222 strain is given by Stanley *et alii* (1953*a*).

TABLE IV.

Antibody Estimations in Pooled Human Serum from North Queensland.

Date of Pools.	Type and Strain of Virus.[1]			
	B1. Conn. 5.	B2. T80.	B3. K137.	B4. S10B.
1. 7.51 to 30. 9.51 ..	−	+	−	+
1.10.51 to 31.12.51 ..	−	+	−	+
1. 1.52 to 31. 3.52 ..	−	+	±	+
1. 4.52 to 30. 6.52 ..	−	+	±	+
1. 7.52 to 30. 9.52 ..	−	+	−	+
1.10.52 to 31.12.52 ..	−	+	−	+
1. 1.53 to 31. 3.53 ..	−	±	−	+
1. 4.53 to 30. 6.53 ..	−	+	±	+
1. 7.53 to 30. 9.53 ..	−	+	−	+
1.10.53 to 31.12.53 ..	−	+	−	+

[1] "+" = 0/8–2/8; "±" = 3/8–6/8; "−" = 7/8–8/8. Numerator, number of mice paralysed; denominator, number of mice inoculated. The sera were tested at a dilution of 1/20.

B697, J699, H700, G703 Strains—Group B, Type 4.

The effects produced in infant mice by each of the B697, J699, H700 and G703 strains were similar. Adaptation was slow compared with that of the M222 strain, and a high mortality was not produced until about the seventh passage.

During the first three passages, signs of illness began to appear about the fifth day after inoculation. General weakness was the most common feature, being observed in approximately half the mice. Emaciation, tremors, panting and paralysis were noted in a smaller proportion. Paralysis was seen mainly in the hind limbs and was a late symptom, appearing between the sixth and ninth days. In the early passages, death occurred in about a quarter of the mice, usually between five and eight days after inoculation. With successive passages, signs appeared earlier. The mortality rate increased and survival time decreased. Often deaths occurred suddenly after only slight manifestations of illness. Paralysis was less often observed. When well established by the ninth passage, the viruses caused the death of all mice within two or three days of inoculation.

The only macroscopic abnormality in the mice, seen occasionally at autopsy, was a necrotic appearance of the fat pads over the scapulæ. In 15 mice examined histologically, the most regular finding was inflammation of fat. Mild focal myositis was commonly seen, and in a small proportion of mice, myocarditis, encephalitis and myelitis. These lesions are characteristic of group B viruses.

Each strain was tested for pathogenicity in weaned mice. Four mice were inoculated intraperitoneally, and four intracerebrally, with infective material. They were quite resistant to the viruses compared with the infant mice. The only abnormal appearance during the observation period of twenty-one days was a slight ruffling of the coat at two or three days. No histological examination of these mice was made. The strains were also tested for pancreatotropic effect, by means of the method described by Howes (1954). Four weaned mice were inoculated intraperitoneally with each strain, and four days later were injected intraperitoneally with trypan blue. The pancreases of all the mice were stained, while those of six uninfected control mice were not.

As a check on the viral nature of the agents, muscle suspensions were filtered through Gradocol membranes of 340-350 mμ average pore diameter. All filtrates proved infective for infant mice.

In Sydney, all four strains were identified as group B, type 4.

W864 Strain.

Two of eight mice inoculated with the fæces of W864 (collected on May 18, 1954) showed retardation of growth at three and seven days respectively. The former recovered, the latter was used for passage. The other six remained well. In the second passage, two mice became paralysed in the hind legs at six days and were sacrificed. The other four were weak at seven to eight days but survived. With continued passage there was a reduction in incubation period and an increase of severity. Paralysis occurred at six to seven days in three of the 12 third-passage mice. In the fourth passage, all 11 mice became weak at three or four days, two died at four days and one became paralysed at six days. In the fifth passage, 15 of 22 mice died within two days.

The limbs of some mice showed mild flaccid paralysis. Usually the mice were more severely affected, and there were contortion and fixation of limbs by the severe muscle lesions. On dissection, these could be seen grossly as large, hard, whitish areas. Histological examination of three mice of the third passage showed widespread and severe destruction of voluntary muscles, but no other lesion.

TABLE V.

Age Distribution of Patients Examined for Coxsackie Virus.

Observation.	Age in Years.					
	0 to 9.	10 to 19.	20 to 29.	30 to 39.	40 to 49.	50 to 59.
Number of patients examined	10	25	20	14	6	4
Number of isolations of Coxsackie virus	2	3	0	2	0	0

Material of the third passage was inoculated intracerebrally into six weaned mice and one brain-to-brain passage was made, but no sign of infection occurred. Howes's test for pancreatotropic effect was performed twice, with material from the fourth and fifth infant mouse passages; in none of the 12 inoculated mice was staining of the pancreas found.

The effects of the W864 virus in mice suggest that it belongs to group A, but its type has not yet been determined. Neutralization tests with hyperimmune antisera prepared against Coxsackie viruses of group A, types 1 to 10, and group B, types 1 to 4, did not reveal any antigenic relationship.

Vitamin E Deficiency.

In the course of these investigations there were two periods during which we were puzzled by the inability to obtain a virus by subinoculation from certain experimental mice which showed features suggesting Coxsackie infection. A few mice had retarded growth and general weakness (a very few had paresis of limbs), and when these were sacrificed some or many muscles were found to be firm, whitish and opaque. However, this appearance of the muscles was noticed more often at the routine

dissection, at twenty-one days, of mice which had shown no symptoms. The nature of these changes became clear when Pappenheimer's (1942) description was found of the muscle necrosis in suckling mice caused by vitamin *E* deficiency.

The mice studied by Pappenheimer were the progeny of mothers fed on a synthetic diet and provided with just sufficient α-tocopherol to permit the birth of living young and the survival of most of them. Muscle lesions developed in 15% during the first thirty-five days of life. The lesions might arise early, but were more frequent and severe during the latter part of the suckling period, in mice that were otherwise healthy. Histological examination revealed hyaline necrosis and fragmentation of muscle fibres, accompanied by interstitial œdema and cellular infiltration. Necrosed fibres were liable to early calcification. Active regeneration of muscle cells followed, and the lesions healed after the mice were weaned, without administration of vitamin. Calcified deposits might persist indefinitely in the muscles.

Our mice behaved very like those of Pappenheimer. However, by the time that we recognized the nature of the changes they had ceased to occur. In retrospect, some points of distinction from Coxsackie myositis have become clear. Mice inoculated with Coxsackie virus when one day old may be expected to become paralysed or die within about two to nine days. Hypovitaminosis should be suspected when muscle damage is found at a later period, particularly in mice with few or no symptoms. When calcification has supervened, the histological picture is distinctive. If muscle lesions not due to Coxsackie infection were to appear early, the clearest indication of their nature would be the failure to reproduce them in passage.

Summary.

Seven strains of Coxsackie virus, representing four types, have been isolated from human fæces in Queensland. The types are group A, type 7, group B, types 3 and 4, and one of group A, not yet defined.

A group A, type 7 strain was isolated from one of seven cases of aseptic meningitis investigated in Brisbane.

A group B, type 3 strain was isolated in January, 1952, from one of five Innisfail cases diagnosed as Bornholm disease, and another group B, type 3 infection, diagnosed serologically, probably arose in the laboratory.

The simultaneous isolation in November, 1953, of four strains of group B, type 4, from 12 cases of "fever" investigated in the Innisfail district, and the failure to isolate this strain throughout the following year, point to an epidemic manner of spread.

The untyped group A virus was obtained from a case of "fever" at Innisfail in May, 1954.

The presence of neutralizing antibody against group B, type 2, and group B, type 4 Coxsackie viruses in many individual sera and in pooled serum suggests that these types have had a wide distribution in Queensland.

Neutralizing antibody may be persistent. In two group B, type 3 infections it was still present three years afterwards; with one group B, type 4 infection, sixteen months afterwards.

By causing muscle necrosis, vitamin *E* deficiency in experimental mice may lead to confusion in Coxsackie investigations.

Acknowledgements.

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CLINICAL OBSERVATIONS ON INFECTIONS WITH INFLUENZA AND COXSACKIE VIRUSES IN NORTH QUEENSLAND, 1953–1954.

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AN epidemic of influenza was in progress when the Field Station of the Queensland Institute of Medical Research commenced operations in Innisfail in July, 1951, and three strains of influenza A virus were isolated (Sinnamon, Pask and McCaffrey, 1952). During an outbreak of Bornholm disease early in 1952, a Coxsackie virus of Group B, type 3, was isolated (Derrick *et alii*, 1956).

In the last quarter of 1953, a widespread outbreak of influenza-like illnesses occurred in the Innisfail-Babinda-Gordonvale area. Its course in Innisfail is depicted in Figure I, which shows the number of patients admitted to the Innisfail District Hospital with the diagnosis of "influenza", "upper respiratory tract infection", "dengue" or "pyrexia of unknown origin". A striking rise in incidence is apparent during November and December, reaching its peak in the week commencing December 6.

Investigation of this epidemic led to the isolation of two infective agents. A virus identified as influenza B was isolated from throat washings from two patients, and a Coxsackie virus typed as group B, type 4, from the fæces of four patients. Examination of paired sera showed antibody response against one or other of these viruses in a number of patients.

Many illnesses in that epidemic showed no specific features, and were difficult to distinguish from the milder cases of leptospirosis seen in the area. Accordingly, during 1954, throat washings, fæces and paired sera were obtained from many "fever" patients and submitted for examination. As a result, a number of cases of influenza A were diagnosed, and a Coxsackie virus (not yet typed) was isolated from one patient.

The clinical and epidemiological features of the patients in whom evidence of influenza or Coxsackie infection was found are described in the present paper, and their significance in the general investigation of North Queensland fevers is discussed.

1953 — Influenza B.

Twelve patients gave definite evidence of influenza B infection; in two the virus was isolated, and in 10 a

diagnostic rise in antibodies was demonstrated between acute and convalescent sera, by either complement-fixation or hæmagglutination-inhibition tests. Four other patients had suggestive evidence, a high titre of antibodies being found in a convalescent serum, while no acute serum was available for testing. The cases in which the virus was isolated are described below.

A., aged eighteen years, cadet engineer, of Innisfail, was admitted to the Innisfail District Hospital on December 17,

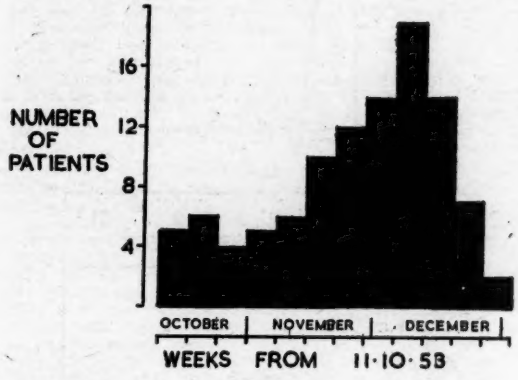

FIGURE I.

Weekly admissions to Innisfail District Hospital of patients with short febrile illness, or upper respiratory tract infections, between October 11, 1953, and January 2, 1954.

1953. He had fallen ill on December 15, with feverishness, and a cough productive of small amounts of sputum, which he noticed was blood-stained on occasions. On examination of the patient, his temperature was 101° F., his pulse rate was 104 per minute, and crepitations were heard at the bases of both lungs. The provisional diagnosis was bronchopneumonia, and he was given 500,000 units of penicillin immediately and 100,000 units every three hours. On

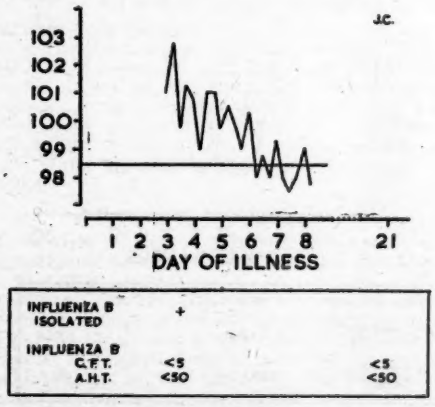

FIGURE II.

Temperature chart of A.; influenza B, 1953.

December 18 an X-ray film of his chest was normal; he complained of a sore throat. The course of his illness is shown in Figure II; his temperature fell by lysis, and he was discharged from hospital on December 23. Influenza B was isolated from throat washings collected on December 18. Convalescent serum taken on the twenty-first day did not show any antibodies against that virus to complement-fixation or hæmagglutination-inhibition tests.

B., aged fourteen years, a cane-farm labourer, of Moresby, near Innisfail, was admitted to the Innisfail District Hospital on December 18, 1953. His illness had commenced on December 16 with cough, sore throat and feverishness. On examination of the patient, his temperature was 102·5° F., his pharynx appeared reddened, and his chest was normal. The provisional diagnosis was "upper respiratory tract infection"; he was given penicillin, 100,000 units every three hours. The course of his illness is shown in Figure III. He was discharged from hospital, well, on December 22. Throat washings were taken on December 18, and influenza B virus was later isolated from them. Examination of a specimen of late convalescent serum revealed a moderate titre of complement-fixing antibodies against that virus.

The diagnosis in the following case depended on the development of anti-hæmagglutinins in convalescence. The clinical features of his illness were less typical of influenza.

C., aged thirty-six years, a canecutter, of Fishery Falls, was admitted to Babinda Hospital on November 12, 1953. He had fallen ill on November 11, with headache, shivering, nausea, and vomiting; he had a dry cough, but no other respiratory symptoms. His doctor noted some palpable inguinal glands, and commenced treatment with penicillin, 400,000 units every four hours, on the provisional diagnosis of leptospirosis. The course of his illness is shown in Figure IV; he remained febrile for three days, and then was afebrile for two days; this period was followed by a further six days of fever. During his second febrile period his axillary, supraclavicular and left inguinal glands were palpable; he had no respiratory signs or symptoms at that time. Anti-hæmagglutinins were not detected in his serum taken on the second day of illness, but were present to a

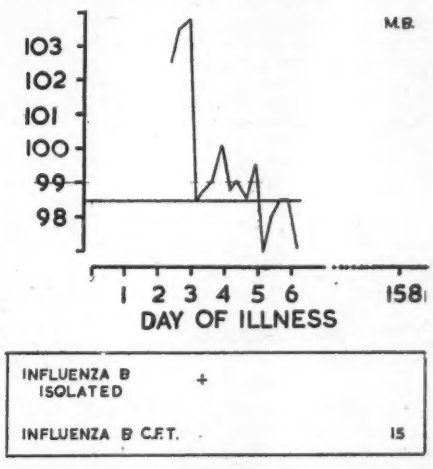

FIGURE III.

Temperature chart of B.; influenza B, 1953.

titre of 1/125 in serum taken on the fifty-sixth day. His serum gave negative results to complement-fixation tests against dengue type 1 antigen.

This biphasic chart was not seen in any other case proven to be influenza, but, as will appear below, it was not uncommon in other cases considered to form part of the epidemic.

Clinical Features.

The signs and symptoms found in the 16 cases may be set out as follows:

Feverishness	10
Headache	11
Stiff neck	1
Backache	5
Cough	11
Sore throat	6
Earache	1
Sore eyes	1
Chest pain	1
Vomiting	5
Lymph gland enlargement	6
Conjunctival injection	3
Faucial injection	5
Abnormal chest signs	4
Albuminuria	1
Rash	1

Average duration of fever: 5·2 days.

It will be noted that cough, sore throat and chest signs were common, but five patients had no evidence of respiratory tract involvement.

One patient had albuminuria and an erythematous rash. His illness was not diagnosed beyond doubt, as no serum was taken in the acute stage. A sample taken on the tenth day had an anti-hæmagglutinin titre of 1/1500 against influenza B.

Figures II, III and IV give examples of the febrile course shown by these patients. The total duration of the primary fever varied from three to eight days, with an average of 5·2 days. Two showed rises in temperature after an afebrile period of twenty-four hours or more; in one case the rise consisted of two readings of 99·4° F. four hours apart, but the other patient (C. above) had a relapse lasting six days.

Case Incidence.

The 16 patients included two women and one child, reflecting the occupational bias in the patients referred to the Field Station. Seven were from Innisfail or its surroundings, five from the Babinda area and three from Gordonvale, and one had travelled from Innisfail to Cairns before his illness. The three patients from Gordonvale fell ill on September 14, October 9 and 12, the Babinda patients

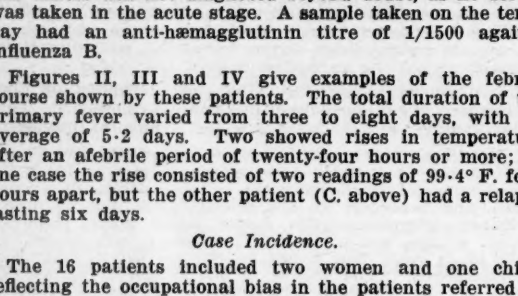

FIGURE IV.
Temperature chart of C.; influenza B, 1953.

on October 11 and 13, November 10, 16 and 20, and the Innisfail patients on October 13 and 15, November 14 and 15 and December 15, 16 and 26. Cases occurred in Innisfail, therefore, over a period of ten weeks.

1953 — Coxsackie Group B, Type 4.

Coxsackie viruses typed as Dalldorf's group B, type 4, were isolated from the fæces of four patients. The serum of three of these developed neutralizing antibody against this type. The serum of 26 other patients who became ill during the period of the epidemic were examined (Table III of Derrick *et alii*, 1956), and two specimens showed a fourfold rise in antibody suggestive of recent Coxsackie group B, type 4 infection.

The histories of the four patients from whom viruses were isolated are as follows.

D. (H700[1]), aged six years, of Coorumbah (ten miles from *Innisfail*), was admitted to the Innisfail District Hospital on November 22, 1953. He had become ill that morning, and was feverish and complaining of abdominal pain. His appetite was poor; he had no urinary or respiratory symptoms. On examination of the patient, his temperature was 103° F., the pharynx appeared reddened, the chest and abdomen were normal, and axillary and inguinal glands were palpable and tender. His ear drums appeared normal. On November 23 his temperature was normal; he had no abdominal pain, but complained of some chest pain. On November 24 his temperature rose to 99·4° F., and he appeared tender to palpation over his liver edge. His white cell count that day was 9000 per cubic millimetre. He had no symptoms on November 25, and was discharged from hospital, well, on the following day.

This patient's chart is shown in Figure V. The fæces from which a virus was isolated were collected on

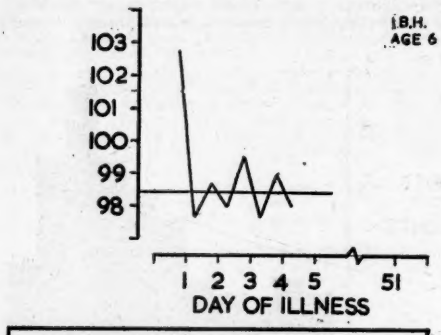

FIGURE V.
Temperature chart of D.; Coxsackie group B, type 4 infection, 1953.

November 24; his serum developed a high titre of neutralizing antibody against the test strain (G703) of Coxsackie virus group B, type 4.

E. (B697), aged twelve years, of Innisfail, was admitted to the Innisfail District Hospital on November 23, 1953. The onset of illness was on November 21, when he was feverish;

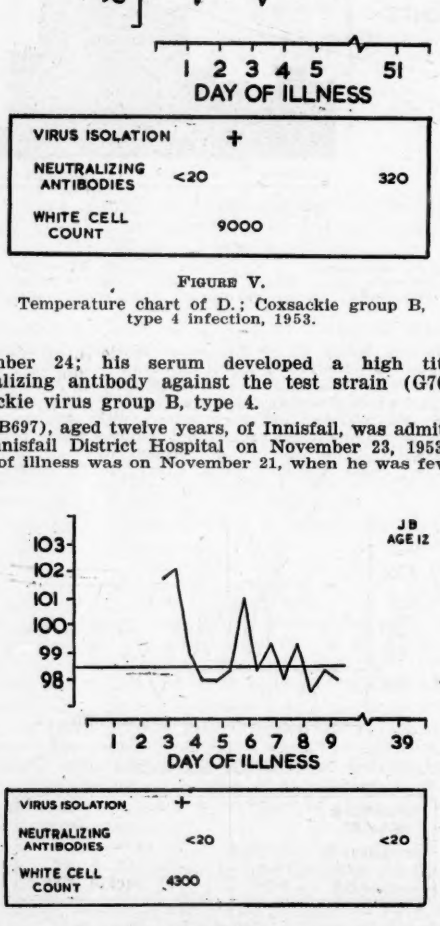

FIGURE VI.
Temperature chart of E.; Coxsackie group B, type 4 infection, 1953.

on November 22 he complained of headache, sore throat and a cough. On examination of the patient, his temperature was 101·8° F., and his pulse rate was 126 per minute; otherwise no abnormalities were found on physical examination. He was given penicillin, 100,000 units every three hours. On November 24 he was brighter; his white cell count was 4300 per cubic millimetre. On November 25 he was afebrile, but he had a recurrence of fever for the next three days. Administration of penicillin ceased on November 29, and he was discharged from hospital on November 30. This patient's chart is shown in Figure VI. A Coxsackie virus,

[1] This and subsequent code numbers will permit identification of the patients in the paper of Derrick *et alii* (1956).

group B, type 4, was isolated from fæces collected on November 24; however, no neutralizing antibody against his own virus or against the G703 strain could be demonstrated in his convalescent serum. Serological tests for influenza and dengue also gave negative resluts.

F. (J699), aged thirty-four years, a housewife, of Wangan, near Innisfail, was admitted to the Innisfail District Hospital on November 20, 1953. Her illness had commenced on November 17, with headache, nausea and sore eyes. On November 18 she was anorexic and had severe headache and backache. She was examined by her physician, who recorded her temperature as 103·8° F. On November 19 she felt rather better, but on November 20 she had severe headache again, and was admitted to hospital. On examination of the patient then, her temperature was 101·8° F. and her conjunctivæ were injected, but no other abnormalities were found. She recovered quickly after her admission to hospital and was discharged on November 24. A virus was isolated from fæces collected on November 23, a rise in neutralizing antibody against the G703 strain was demonstrated in her serum.

G. (G703), aged thirty-seven years, a cane-farmer, of Little Mulgrave, was admitted to the Gordonvale Memorial Hospital on November 17, 1953. He had fallen ill on November 16 with headache, weakness, shivering and anorexia; he had no respiratory or urinary symptoms. On examination of the patient, his temperature was 102·4° F., his right axillary glands were palpable and tender, and firm, non-tender glands could be felt in his groins. His abdomen was slightly tender under the right costal margin, and his eyes were injected. The possibility of leptospirosis was considered; however, he made a rapid recovery without antibiotics, being afebrile on November 19. His chart is shown in Figure VII. A virus was isolated from fæces collected on November 19, and he developed a high titre of neutralizing antibody against that strain. Attempted culture of leptospiræ from the blood gave negative results, and agglutination tests against the Queensland serotypes of leptospiræ showed only residual antibodies from a proven attack of leptospirosis nine months earlier.

Thus, the four patients had short febrile illnesses without specific features; they could not be distinguished clinically from the influenza group, although the respiratory tract symptoms (cough and sore throat) were seen less frequently. None of the patients had myalgia severe enough to suggest Bornholm disease. However, it is noteworthy that abdominal pain was the presenting symptom in one case (D. above). Similarly, none had back or neck stiffness that would suggest aseptic meningitis.

Two of the four patients were children, and one was a woman. Three from the Innisfail district fell ill on November 17, 21 and 22, and one from Little Mulgrave (near Gordonvale) on November 16. The occurrence of their illnesses in one week is simply due to the fact that the 12 samples of fæces examined for virus were all collected within a week. However, the serological tests covered a wider interval, without revealing cases outside that period.

The Nature of the 1953 Epidemic.

In all, 60 cases considered to belong to the same epidemic were investigated more or less fully in the last quarter of 1953. Attempts were made to isolate leptospiræ, rickettsiæ, and Coxsackie, influenza and other viruses from representative groups of patients. Serological tests were carried out, as far as serum was available, for evidence of leptospirosis, the rickettsioses, brucellosis and typhoid and paratyphoid fevers, as well as for Coxsackie infection and influenza.

It is clear that influenza B made a considerable contribution to the epidemic. The virus was isolated from the throat of two of the five patients tested; 10 of 34 whose paired sera were examined showed a significant rise in antibody. The proportion of cases of influenza was probably higher than this, as the occurrence of clinically typical but serologically negative cases is well recognized (Stuart-Harris, 1953; Harding *et alii*, 1954), and is exemplified in the present series by patient A., whose case is described above.

It is more difficult to assess the part played by the Coxsackie virus in the epidemic. The virus was isolated from the fæces in four of 12 cases, and at least a fourfold increase in neutralizing antibody was found in the serum of five of 27 patients. There are many references to the finding of group B Coxsackie viruses in association with a short febrile illness, and if one could assume that the illness that occurred in these North Queensland patients at the time of the Coxsackie virus infection was due to that infection, these figures would give an approximate indication of the part played by this virus in the epidemic. However, it is arguable that the virus infection was simply coincidental, for there was no clear distinction between the clinical features of cases with and without laboratory evidence of Coxsackie infection. At the most, it would appear that the part played by the Coxsackie virus in the epidemic was a relatively minor one.

An outbreak of dengue in North Queensland, apparently beginning in Townsville at the end of 1953 (O'Connor *et alii*, 1955), added another facet to the fever position. Although clinically typical cases did not appear in the Innisfail district until March, 1954, the question arises whether dengue may have been responsible for some of the Innisfail cases in 1953. Twenty-nine paired Innisfail sera were therefore tested for complement-fixing antibodies against dengue type 1, Murray Valley encephalitis (MVE), or West Nile virus. These three give considerable, though inconstant, cross-reaction. A fourfold rise in antibody was found in one patient only, and her clinical features were not typical of dengue. The contribution of the dengue group to the epidemic, if any, was slight.

A final diagnosis was not made in many of the epidemic cases. It was not practicable to carry out the full range of tests in all cases, and some may well have escaped the relevant test. However, there were a number that remained undiagnosed in spite of full investigation. This group did not present any clinical or epidemiological features that would permit them to be distinguished from those providing evidence of influenza or Coxsackie infection. One illustrative case, with clinical features typical of the outbreak, is described below.

H., aged eighteen years, a labourer, of Bartle Frere, was admitted to the Babinda District Hospital on November 4, 1953. His illness had begun on November 2, with headache, pains in the legs, sore throat, and occasional cough. He had some dysuria, but no frequency of micturition, and developed a backache after his admission to hospital. On examination of the patient, his temperature was 99° F., his pharynx was reddened, axillary and inguinal lymph nodes were palpable, the chest was clear, and no tenderness was found over the liver or spleen. On November 5 he had large, tender cervical and axillary glands, and his eyes were red and sore. His temperature rose to 103·6° F. His subsequent course is shown in Figure VIII; the temperature fell to normal by the eighth day of illness, and he was discharged from hospital three days later. Culture of leptospiræ from the blood was attempted and inoculation of blood into weaned mice was performed, with negative results. The patient had no serological evidence of infection with the indigenous fevers, influenza, Coxsackie group B, types 2 or 4, or the dengue group (with the use of MVE antigen).

It may be concluded that the epidemic was largely due to influenza B. A Coxsackie virus was circulating at the same time, but its contribution to the illnesses is difficult to assess. Viruses of the dengue group played a very slight part, if any. There is no clinical or laboratory evidence that other infective agents were present, but the possibility that some of the respiratory infections were due to the A.P.C. group of viruses has not been excluded by the tests employed.

1954 — Influenza A.

Between May and September, 1954, an epidemic of influenza A passed through the coastal belt of North Queensland from Innisfail to Mossman. Eighteen of the patients investigated by the Field Station during that time were finally diagnosed as suffering from influenza A. All showed a diagnostic antibody response, and influenza virus was isolated from the throat washings of five.

Five of the 18 patients were chosen for investigation as suffering from typical influenza. The remainder were investigated after they had been referred by their attending physicians with the diagnosis of "pyrexia of unknown origin", although a provisional diagnosis of influenza had been made in some of these cases. This is therefore a

selected series, and the clinical features and some of the epidemiological features cannot be taken as typical of the epidemic as a whole.

The difficulties in diagnosis that arose are illustrated in the following histories:

I., aged nineteen years, a cane-cutter, of Daradgee, was admitted to the Innisfail District Hospital on July 14, 1954. He gave a history of feverishness, headache and backache of twenty-four hours' duration, and admitted that he had a cough and a slightly sore throat. He had been cutting cane during wet weather; one member of his gang had caught leptospirosis in the same area in the previous month, and another (I.'s brother) had been admitted to hospital on the previous day with an illness diagnosed clinically as leptospirosis (which it was ultimately proved to be). On examination of the patient, his temperature was 102° F., his eyes were not injected, his chest and abdomen appeared normal, a few small glands palpable in his groins were not considered significant, his fauces were reddened, and his uvula was œdematous. He was considered to have influenza, although with no degree of certainty, and was given symptomatic treatment only. On the following day he was still febrile, and his white cells numbered 5200 per cubic millimetre. During the afternoon of July 16 he had an episode of severe abdominal pain and vomiting; it terminated after some hours, leaving him with epigastric tenderness. The following day he felt somewhat better, but his temperature reached 100° F. He was by then very discontented with his lack of treatment and made several requests for "the needle". Fortunately, he was well and afebrile on the

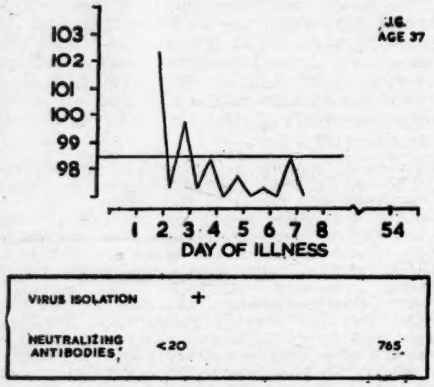

FIGURE VII.
Temperature chart of G.; Coxsackie group B, type 4 infection, 1953.

following day, and was discharged from hospital on July 20. His temperature chart is shown in Figure IX. Influenza A virus was isolated from his throat washings, and a rise in complement-fixing antibodies was demonstrated in his serum. He showed no serological or cultural evidence of leptospirosis.

J., aged forty-one years, a timber cutter, of Innisfail, was admitted to the Innisfail District Hospital on July 21, 1954. He had fallen ill on July 20, with headache, feverishness, a cough, sore eyes, epigastric pain and backache. On examination of the patient, his eyes were red and injected, the right axillary lymph glands were palpable and tender, his temperature was 104° F., and examination of his chest and abdomen revealed no abnormality. He had been engaged in timber work around a sawmill and in the scrub during wet weather. He was provisionally diagnosed as suffering from leptospirosis, and was indeed included in a series for therapeutic trial of erythromycin. The course of his illness is shown in Figure X. He was still ill on July 22, with severe headache, and lay avoiding the light. His white cells numbered 4300 per cubic millimetre. On July 23 his temperature fell to normal, and he felt somewhat better. He was discharged from hospital on July 26. Attempted culture of leptospiræ from the blood gave negative results, and he showed no serological evidence of leptospirosis in his convalescent serum. However, there was a diagnostic rise in complement-fixing antibodies against influenza A.

The following patient had a biphasic temperature chart, and a striking generalized enlargement of lymph glands. He developed anti-hæmagglutinins, but no complement-fixing antibodies.

K., aged ten years, a school child, of Moresby, near Innisfail, was admitted to the Innisfail District Hospital on August 2, 1954. He had fallen ill on August 1, with headache, pains in his knees, feverishness, a running nose, cough, and some soreness of his throat. He vomited on one occasion, but his appetite remained fair. On examination of the patient, his temperature was 102° F., his eyes were injected, his throat, chest and abdomen were normal. Large lymph glands were present in his groins, axillae and posterior triangles, and in his tonsillar and epitrochlear groups. On August 3 his temperature reached 104° F., his white cell count was 8000 per cubic millimetre, and treatment with penicillin was commenced. His subsequent course is shown

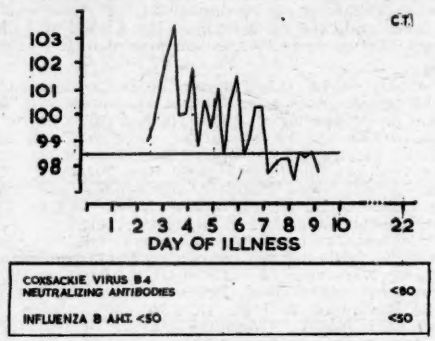

FIGURE VIII.
Temperature chart of H.; undiagnosed case from 1953 epidemic.

in Figure XI; he had a biphasic temperature chart, and was finally discharged from hospital on August 9. At that time his glands were smaller but still palpable. He had a diagnostic rise in antihæmagglutinins, but no complement-fixing antibodies were found. Investigations for other infective agents (leptospirosis, typhus, Coxsackie infections, dengue and Murray Valley encephalitis) all gave negative results

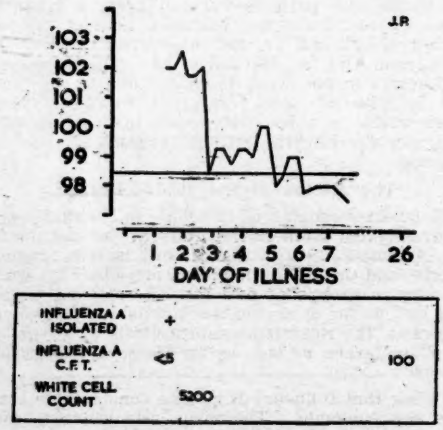

FIGURE IX.
Temperature chart of I.; influenza A, 1954.

Clinical Features.

The signs and symptoms recorded in these 18 cases may be set out as follows:

Feverishness	10
Headache	14
Backache	7
Muscle pains	3
Cough	13

Sore throat	7
Nasal congestion	3
Sore eyes	8
Vomiting	8
Diarrhœa	1
Lymph gland enlargement	10
Conjunctival injection	8
Faucial injection	8
Abnormal chest signs	8
Liver tenderness	2

Average duration of fever: 4·9 days.

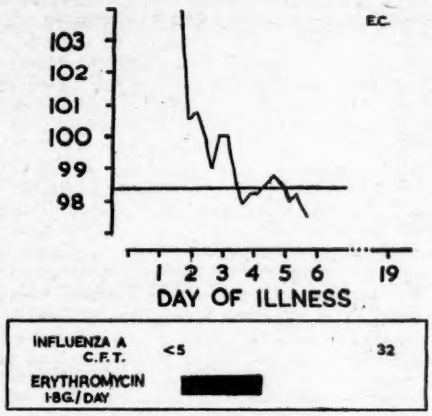

FIGURE X.
Temperature chart of J.; influenza A, 1954.

The febrile period lasted between three and nine days, with an average of 4·9 days. In three, a temperature over 99° F. was recorded on one or more occasions after the patient had been afebrile for at least twenty-four hours, but the rise was short and of doubtful significance. The highest temperature recorded was 105° F.; one patient remained afebrile in hospital; 13 patients had maximum temperatures of 102° F. or higher.

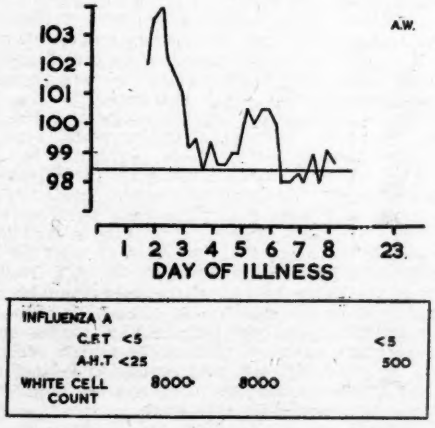

FIGURE XI.
Temperature chart of K.; influenza A, 1954.

Case Incidence.

The 18 patients include only one woman and one child; the geographical distribution of the cases ranged from Moresby, eight miles south of Innisfail, to Julatten. The first case recognized had its onset on May 9, 1954, and the last on September 11, 1954. Babinda was affected in May, but Innisfail not until the end of June. The highest incidence appeared to occur in July, when 10 cases were diagnosed. In three cases in which the diagnosis was made on clinical grounds, negative serological results were obtained. McLean *et alii* (1955) similarly obtained negative serological results in only five of 55 cases in the 1954 influenza A epidemic in Victoria.

1954 — Coxsackie Virus Investigations.

During 1954, attempts were made to isolate Coxsackie viruses from the fæces of 43 patients. One virus was isolated, but its relationship to the illness under investigation has not been established. Its behaviour in mice suggested that it belonged to Dalldorf's group A, but it showed no serological relationship to any of the known types within that group (Derrick *et alii*, 1956). The following is the clinical history of the patient from whom the virus was isolated:

L. (W864), aged six years, schoolgirl, of Flying Fish Point (near Innisfail), was admitted to the Innisfail District Hospital on May 17, 1954. Her mother gave a vague history that the child had been irritable and feverish for two days. On examination, she had a macular rash on the trunk and pronounced lymph-node enlargement involving her inguinal, axillary, cervical and epitrochlear groups. Her temperature was 101·4° F. No abnormalities were found in her chest, abdomen, throat or ears. A provisional diagnosis of "dengue" was made, and she was treated symptomatically. Her course is shown in Figure XII. On May 18 her rash was fading,

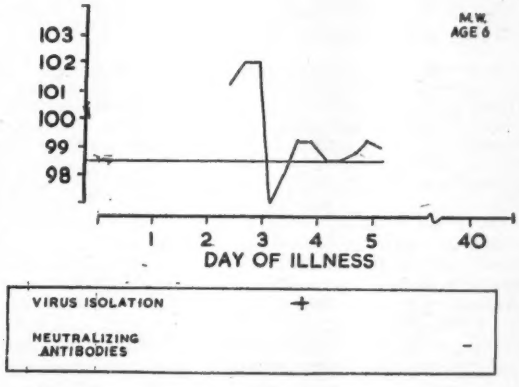

FIGURE XII.
Temperature chart of L., from whom a Coxsackie virus

and she was discharged from hospital on May 20. Fæces were collected on May 18, and transported to Brisbane on dry ice. The patient's convalescent serum contained no antibody against the virus isolated. Serological tests against influenza A and B, dengue type 1, MVE, and West Nile antigens gave negative results. However, the patient's clinical condition resembled that of a number of others from Flying Fish Point examined during 1954, some of whom were proved to have dengue.

While it was not possible to have many 1954 sera tested for evidence of Coxsackie infection, the almost negative results from attempts at isolation suggest that Coxsackie viruses were not prevalent in North Queensland during that year.

Discussion.

Influenza.

Both the influenza epidemics reported here formed part of larger epidemics that involved at least the whole of eastern Australia. White *et alii* (1954) described an epidemic of influenza B in Victoria between July and September, 1953. Influenza B virus was isolated in Brisbane in October by Mrs. V. M. Macdonald, and was apparently responsible for an epidemic there in October-November. Epidemics occurred in Rockhampton and Townsville between September and December.

McLean *et alii* (1955) reported an epidemic of influenza A in Victoria in April-May-June, 1954. The Melbourne outbreak commenced in May, although cases were recognized at Corryong three weeks earlier. The Brisbane outbreak commenced in May, where a strain of influenza A was isolated by Miss I. A. Brown from lung collected *post mortem* on May 10, 1954.

Therefore, while over two months separated the first cases of influenza B in Victoria and Queensland, influenza A appeared in Melbourne, Brisbane and Babinda at almost the same time. This is broadly in keeping with Stuart-Harris's statement (1955) that influenza B is characterized by a series of localized outbreaks, and influenza A by a tendency to sweep across the country. However, it is puzzling that Innisfail, 20 miles from Babinda, should not have had its influenza A outbreak until seven weeks after the first case had been diagnosed in Babinda.

No significant difference was noticed between the clinical severity of the two epidemics. In both there were some patients who were very ill and had to be admitted to hospital, and a widespread incidence of trivial and moderately severe cases among the community, some subjects being ambulant. There was no evidence for the suggestion (Editorial, 1955) that influenza A causes a more severe illness than influenza B.

It is of interest that the influenza B epidemic continued into summer, until about the end of December. There was also an isolated case at the end of January, 1954. Horsfall (1952) mentioned that an epidemic might develop at any time of the year, "especially in the tropics where epidemics have occurred during the summer months".

Coxsackie Group B, Type 4 Infections.

The type of Coxsackie virus isolated in North Queensland in 1953 (Dalldorf's group B, type 4) has been recorded on a number of occasions, and from widely separated localities. Melnick (1953) isolated the Texas-13 strain from flies, and Cheever *et alii* (1950) the Powers strain from a patient in Massachusetts diagnosed as suffering from non-paralytic poliomyelitis. Howes (1951) isolated the type from a patient with pleurodynia at Adelaide, South Australia, and Stanley *et alii* (1953) from Sydney sewage. Patz *et alii* (1953) found it in association with an outbreak of Bornholm disease in South Africa.

Relation to the "Pyrexia of Unknown Origin" Problem in North Queensland.

Quite a high proportion of the patients reported in this paper were initially diagnosed, by their own doctor or by the writer, as suffering from "pyrexia of unknown origin", "leptospirosis", and even, in several cases, "scrub typhus". Leptospirosis, treated with penicillin, is seen in North Queensland as a febrile illness with an average duration of 5·2 days; respiratory signs and symptoms are not uncommon, and jaundice is rare (Doherty, 1955). Its diagnosis from influenza without respiratory symptoms can therefore be difficult. The 1953 outbreak of influenza occurred during a dry period, when few cases of leptospirosis presented; errors in diagnosis were limited to the early days of the epidemic, before its presence was generally realized. However, the 1954 outbreak coincided with a period of moderately high incidence of leptospirosis, and a confident diagnosis was often not possible, especially in relation to patients whose occupation exposed them to leptospiræ.

This work must be viewed against the general background of the fever investigations in North Queensland. The isolation of new serotypes of leptospiræ, and the use of mouse inoculation as a routine procedure for scrub typhus diagnosis, have lowered the proportion of cases remaining undiagnosed. The largest element remaining appears to be short, benign fever of two to five days' duration. That so many of these fevers during 1954 should have proved to be influenza emphasizes the care that must be taken to exclude known diseases before postulating the presence of further new infective agents.

Summary.

1. An outbreak of short febrile illnesses in North Queensland in late 1953 was largely due to influenza B. There were some infections with a Coxsackie virus of group B, type 4 (Dalldorf).

2. In the cases diagnosed as influenza B the average duration of fever was 5·2 days; cough and sore throat were common symptoms, but in several there was no evidence of respiratory tract involvement.

3. The four patients from whom Coxsackie group B, type 4 virus was isolated had an average duration of fever of 4·7 days; none had severe myalgia or signs of aseptic meningitis.

4. A number of patients, whose clinical features suggested that they belonged to the epidemic, gave negative results to investigation. However, no evidence was obtained that any hitherto undescribed agent had caused their infections.

5. Eighteen patients with influenza A were studied during an epidemic in 1954; a number of these had no respiratory signs or symptoms. The average duration of fever was 4·9 days.

6. The influenza epidemics of 1953 and 1954 can be correlated with outbreaks in southern Australia in those years. No significant difference was observed between the clinical severity of influenza B and that of influenza A.

7. A Coxsackie virus of group A was isolated from one of 43 patients examined during 1954. It was probably unrelated to the patient's illness. This scanty result, compared with 1953, suggests that Coxsackie infections may appear in North Queensland only in epidemics.

8. The cases reported are of importance in the investigation of North Queensland fevers, in that some of the influenza and presumed Coxsackie infections closely resembled mild cases of leptospirosis. Difficulties in differential diagnosis were encountered especially during the 1954 influenza A epidemic, which coincided with a period of moderate incidence of leptospirosis.

Acknowledgements.

The Coxsackie virus isolations were carried out by Mr. J. Pope, influenza virus isolations by Mrs. V. Macdonald and Miss I. A. Brown, and MVE complement-fixation tests by Mr. C. Ludford, all of the Queensland Institute of Medical Research. Coxsackie viruses were typed and Coxsackie neutralization tests performed by Dr. N. F. Stanley, of the Institute of Epidemiology and Preventive Medicine, Sydney. Influenza viruses were typed and influenza complement-fixation tests performed by Mr. J. White, of the Epidemiological Research Unit, Fairfield Hospital, Melbourne. Influenza hæmagglutination-inhibition, dengue, MVE, and West Nile virus tests were carried out by Dr. J. L. O'Connor and Dr. L. C. Rowan, of the Commonwealth Serum Laboratories. Serological tests for leptospirosis *et cetera* were carried out by Dr. J. I. Tonge and the staff of the Laboratory of Microbiology and Pathology, Department of Health and Home Affairs, Brisbane. The writer wishes to thank the practitioners of the area for their cooperation, the Medical Superintendents of Innisfail Hospital, Babinda Hospital and Gordonvale Hospital for permission to publish case histories, and Dr. E. H. Derrick for his generous help with this work.

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EXPERIMENTAL USE OF RESERPINE IN THE TREATMENT OF TREMOR-RIGIDITY SYNDROMES.

By DAVID R. MORGAN,
Parramatta.

DURING the treatment of psychotic patients with chlorpromazine I have noticed, as have observers overseas, that some will develop an appearance which resembles Parkinsonism, but which upon examination is not quite typical of that disease; for although the tremor is obvious, there is little evidence of rigidity.

The patients usually present a typical appearance—slightly stooped, slow in their movements, with a shuffling gait and absence of arm-swinging whilst walking, mask-like features, eyes directed downwards and straight before them, and at times profuse and distressing salivation, the saliva being often thick and ropy.

At the same time they develop a pronounced rhythmical alternating tremor of the upper extremities, but not quite the typical "pill-rolling" tremor of Parkinsonism; nor do they exhibit the rigidity usual in that syndrome. However, some other observers have noted a "cog-wheel rigidity" in patients receiving 60 milligrammes (and more) of reserpine per day.

I have had the same state develop in patients during reserpine therapy, and in the early stages of my use of these drugs the development of these Parkinson-like features caused me some disquiet. However, I quickly discovered that by giving these patients an anti-Parkinsonism drug, such as ethopropazine hydrochloride ("Lysivane"), 150 milligrammes daily in three divided doses, the extrapyramidal symptoms were controlled without reducing the dosage of either chlorpromazine or reserpine. Of course, reducing the dosage will achieve the same result in this respect, but that defeats the original purpose of its administration. I have come to the conclusion that the development of these symptoms is no cause for alarm, and this too is the opinion of overseas observers.

Recently in the *Annals of the New York Academy of Sciences* was published a symposium (1955) on the use of reserpine with some reference to chlorpromazine, and one of the articles, by Franco Rinaldi and Harold E. Himwich (page 27), provides the basis for an explanation of this drug-induced tremor, which I will briefly summarize, as it is on this theoretical basis that I have used reserpine in an attempt to ameliorate the symptoms of both senile Parkinsonism and Huntington's chorea.

Rinaldi and Himwich, by placing electrodes within the brain tissue, recorded electroencephalograms from different nerve nuclei and the manner in which their electrical activity is influenced by reserpine, and it was shown that this drug induces a continuous pattern of alertness and stimulates rather than depresses the brain-stem reticular formation or M.D.A.S. (meso-diencephalic activating system).

The M.D.A.S. includes the reticular formation of the brain-stem tegmentum of Moruzzi and Magoun (1949) and the thalamic diffuse projection system of Jasper (1949) which is in the mid-brain at the approximate level of the anterior quadrigeminal bodies. Small amounts of acetylcholine produce an alerting response in the mid-brain reticular formation, whilst atropine paralyses the alerting mechanism.

Di-isopropylflurophosphate (D.P.F.), a powerful anticholinesterase agent, produces a long-lasting pattern of alertness. Barbiturates in increasing doses produce an electrical pattern characteristic of sleep with depression of the M.D.A.S.

It is shown that atropine paralyses the M.D.A.S., but is not an hypnotic; so that quiescence of the M.D.A.S. is part of the pattern of sleep, but it can also occur in the absence of sleep. Conversely, hyperactivity of the M.D.A.S. is part of the pattern of alertness, but can occur without insomnia; and thus it is that reserpine, which stimulates the M.D.A.S., does not produce insomnia.

The brain-stem reticular formation plays an important part in the genesis of the extrapyramidal symptoms of Parkinsonism, and this probably accounts for the fact that large doses of reserpine, which stimulate the M.D.A.S., produce a rhythmically alternating tremor.

It is of interest to note that the most effective anti-Parkinsonism drugs are (like atropine) the most powerful depressants of the brain-stem reticular formation.

Reserpine also causes an increase in cortical inhibition of diencephalic structures, which is probably explained by the fact that it facilitates synaptic transmission. Reserpine also antagonizes the "sham rage" states in appropriately prepared cats, but the presence of the cortex is necessary to achieve a full reserpine effect in these animals.

It was with the above theoretical concepts in mind that I examined the possibility that reserpine might be of value in the treatment of degenerative arteriosclerotic Parkinsonism (and possibly of Huntington's chorea), for owing to degenerative changes in cortical neurons there is a progressive decrease in cortical inhibition of the brain-stem reticular formation, leading to unopposed activity of the M.D.A.S. and a gradual appearance of Parkinsonism symptoms.

Now, as reserpine facilitates cortical inhibition of the M.D.A.S., I anticipated that, in doses smaller than those which might of themselves produce Parkinsonian symptoms in young people, reserpine might in certain cases produce an ameliorating effect upon the symptoms of early Parkinsonism and possibly also of Huntington's chorea, and even perhaps upon spasticity caused by cortical damage.

As it turns out, my results have been equivocal but certainly encouraging, and only one patient in my small series of cases has been made worse, whilst several have shown a tremendous improvement.

The patient (No. 1 in Table I) who became worse has a pronounced hysterical element in her personality make-up; and when she became destructive and made a theatrical gesture at a suicidal attempt, administration of reserpine was discontinued.

I had at the time only 11 patients under my care exhibiting choreiform or rhythmically alternating tremors, of whom the six suffering clinically from Parkinsonism were receiving daily doses of "Lysivane".

Reserpine was administered to the patients in a dosage of one milligramme three times a day, with the results shown in Table I.

As will be seen from this table, seven patients showed either a small improvement or none at all, and three patients were moderately or greatly improved. One patient (No. 5), who was confined to bed, completely unable to carry on a coherent conversation or to feed herself, was so much improved that she was able to care for herself and talk freely; she then proceeded on leave to her family, who previously had found her nursing care an insoluble problem.

TABLE I.

Effect of Administration of Reserpine.

Patient.	Diagnosis.	Age. (Years.)	Degree of Change.
1	Congenital choreoathetosis.	43	Worse.
2	Huntington's chorea.	46	Nil.
3	Huntington's chorea.	54	Small improvement.
4	Huntington's chorea.	56	Small improvement.
5	Senile arteriosclerotic Parkinsonism.	66	Great improvement.
6	Senile arteriosclerotic Parkinsonism.	65	Moderate improvement.
7	Senile arteriosclerotic Parkinsonism.	61	Moderate improvement.
8	Senile arteriosclerotic Parkinsonism.	67	Small improvement.
9	Senile arteriosclerotic Parkinsonism.	65	Nil.
10	Post - encephalitic Parkinsonism.	48	Small improvement.
11	Post - encephalitic choreoathetosis.	44	Nil.

The four patients who showed small improvement with one exception claimed that they could perform simple tasks with greater freedom, and this was the opinion also of the nursing staff.

It would be unwise to make any sweeping claims on this small sample, which can have no real statistical significance, but I do consider the results to be most interesting and I believe that they warrant a further investigation into the use of reserpine in the treatment of states in which there is a decrease of cortical inhibition with a resulting appearance of tremor, rigidity and spasticity.

I wonder if spastic paraplegia-diplegia might respond in the same way.

G. Jacob and N. Kline (1955) have recorded a dramatic response in a case of Huntington's chorea in a physician treated with reserpine, which is of interest in relation to the above; although in my small series the response of patients suffering from Huntington's chorea has not been encouraging.

Acknowledgements.

My thanks are due to Dr. Eric T. Hilliard, Medical Superintendent of the Mental Hospital, Parramatta, for his continuing interest in the use of these new drugs.

This paper is published with the permission of Dr. Donald S. Fraser, Inspector-General of Mental Hospitals, Division of Mental Hygiene, New South Wales Department of Public Health.

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Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Handbook of Ophthalmology", by Humphrey Neame, F.R.C.S., and F. A. Williamson-Noble, F.R.C.S. Eighth edition; 1956. London: J. and A. Churchill, Limited. 6" x 9¼", pp. 371, with many illustrations. Price: 30s.

The first edition appeared in 1927.

"Gullan's Theory and Practice of Nursing", by M. A. Gullan, revised by Marion E. Gould, D.N. Seventh edition; 1956. London: H. K. Lewis and Company, Limited. 5½" x 8½", pp. 309, with illustrations. Price: 18s.

The first edition was published in 1920.

"A Textbook of General Physiology", by Philip H. Mitchell; Fifth Edition, 1956. London, Toronto and New York: McGraw-Hill Book Company, Inc. 9" x 6", pp. 892, with illustrations. Price: $10.50.

Stress is laid on general principles, and basic physiological processes are explained in terms of the physical and chemical processes which underlie them.

"The Practice of Medicine", edited by John S. Richardson, M.V.O., M.A., M.D. (Cantab.), F.R.C.P.; 1956. London: J. and A. Churchill, Limited. 9" x 6½", pp. 1084, with many illustrations. Price: 40s.

The 14 contributors are teachers of medicine in London hospitals; the book is primarily intended for undergraduate students.

"Practical Urology: Case Comments and Late Results", by Alex E. Roche, M.A., M.D., M.Ch. (Camb.), F.R.C.S. (Eng.); 1956. London: H. K. Lewis and Co., Limited. 8½" x 5½", pp. 270, with 132 illustrations. Price: £1 15s.

The book consists of typical and atypical case histories and when possible the after-history.

"Principles of Embryology", by C. H. Waddington, Sc.D., F.R.S.; 1956. London: George Allen and Unwin, Limited. 9½" x 6", pp. 520, with many illustrations. Price: £2 5s.

The author has tried to provide a text-book for students specializing in the subject and to meet the needs of research workers in other branches of biology.

"Chest X-ray Diagnosis", by Max Ritvo, M.D.; Second Edition; 1956. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10" x 7", pp. 640, with 427 illustrations. Price: £8 16s.

Deals with the criteria on the basis of which the diagnosis of disease of the lungs and heart can be established by radiological methods.

"An Outline of the Cytology and Pathology of the Reticular Tissue", by A. H. E. Marshall, with a chapter on the Lipidoses by J. B. Cavanagh; 1956. Edinburgh and London: Oliver and Boyd. 9" x 6", pp. 284, with many illustrations. Price: 42s.

This book is intended, not to give a detailed account of the subject, but to provide an outline likely to be of value to the diagnostic pathologist or to those requiring an introduction to the subject.

The Medical Journal of Australia

SATURDAY, JUNE 9, 1956.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words of phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

MEDICAL PROGRESS IN WESTERN AUSTRALIA.

THE address by Dr. B. W. Buttsworth, the Retiring President of the Western Australian Branch of the British Medical Association, delivered at the annual meeting of the Branch on March 17, 1956, and published in this issue, is worthy of note for two reasons. In the first place, the Branch has made determined progress in the acquisition and equipment of a permanent home for itself. The old Branch offices were in the heart of the city, they were cramped, and members often experienced parking difficulties when they had to gather there on their lawful Branch occasions. The offices have been removed to a building acquired some time ago in King's Park Road, in a pleasant situation, not too far from consulting room and city activities, yet freer from the restrictions of the older premises. A full-time secretary has been appointed and we read that he has taken up his duties with ability and enthusiasm. This means, of course, that a long association with Neilson Hancock and Hugh Hancock, his son, who acted as part-time secretaries for a combined period of twenty-eight years, has been severed. Incidentally, the editorial staff of this journal has been much indebted to both father and son for much willing cooperation and for many kindnesses over the years. The Branch intends to put up a new building on its site in King's Park Road. Dr. Buttsworth comments that the venture will succeed just as surely as Western Australia "continues to grow in population and wealth". Of this there is no possible doubt whatever. The medical profession of Western Australia has shown that it can carry out any project to which it puts its mind, and this brings us to our second point—the success of the Western Australian appeal to its people for funds to erect their own medical school. A total population of 639,716 persons subscribed no less than £556,000. Of this amount, £414,000 was in cash. The achievement is the more notable because the target originally aimed at was £400,000. To the total the members of the medical profession gave the magnificent sum of £40,000. It is difficult to discover how many medical practitioners there are in the State; the Register contains the names of many who no longer practise in Western Australia. There are, however, 578 members of the Western Australian Branch of the Association. The point that we wish to make is that a profession which is so united in the matter of the establishment of a medical school should have no difficulty in finding the money to build a home for its own professional association. Dr. Buttsworth's prediction will surely come true.

THE INSTITUTE FOR THE STUDY AND TREATMENT OF DELINQUENCY.

THE Institute for the Study and Treatment of Delinquency is not a large or particularly well known body in Great Britain, but it is one whose aims are entirely admirable. It is by the way largely but not entirely a medical body. The aims are as follows:

(i) To initiate and promote scientific research into the causes and prevention of crime.

(ii) To coordinate and consolidate existing scientific research on the prevention and treatment of delinquency.

(iii) To provide educational and training facilities for students, magistrates, probation officers and teachers in the scientific study of delinquency and crime.

(iv) To secure coordination between all bodies engaged in similar work in all parts of the world, and ultimately to promote an international organization.

(v) To promote discussion and to educate the opinion of the general public on these subjects by publications and by other means.

(vi) To establish observation centres or other auxiliary services for the study and treatment of delinquents.

(vii) To publish a scientific journal of delinquency and books and monographs on the subject.

The annual report of the Institute (it is called I.S.T.D.) for the year July, 1953, to June, 1954, has recently been received. This is a short document and does not contain a great deal of information about the activities of the Institute, but what it does contain is interesting, and it provides an opportunity to consider its objects and their desirability.

The Institute is still a young body, for its second annual general meeting was held so late as November, 1953. Its membership at June 30, 1954, was 463. However, we note that the "first stage of the scientific reorganisation" of the Institute has been completed; this was necessitated by the transfer of the Institute's psychopathic clinic to the control of the National Health Service. The Institute inaugurated a "scientific group" for the discussion of delinquency problems, and regular meetings have been held. The "disciplines" represented in the group are: biological sciences, criminal law and justice, criminology, education,

ethics and moral philosophy, organic medicine, psychiatry, psychoanalysis, psychology, statistics, social work, sociology, treatment of offenders. In the matter of education there is an Education Committee which, *inter alia*, arranged for the holding of a summer school at Oxford on the subject of "The Changing Attitude of Society towards Criminal Responsibility". Week-end courses for psychiatrists have been held and external lectures have been given. At one week-end course the subject of "The Sexual Offender" was chosen; lectures were divided into four sections, and discussions took place. The Institute was one of the local centres for the extension course arranged by the London University Extra-Mural Department for diploma courses. The Institute took an active part in the Fourth International Course in Criminology. One of the objects of the Institute is the development of an international organization. It is stated that this aim has been met by the development of the International Society of Criminology, which was to hold its third congress in London in September, 1955. The hope is expressed that the Institute will "look forward to increasing activity, particularly in the direction of trying to knit together the various branches of delinquency work" in Britain. At present much work is done in isolation and there is a good deal of overlapping and wastage of effort. With all this effort, which all will agree to be commendable, the Institute lacks funds and is constantly faced with the possibility that it may have to close down. The Government would be foolish to allow this to happen.

This short account of the activities of the Institute for the Study and Treatment of Delinquency has been assembled from its somewhat meagre report in order to present to Australian psychiatrists and practitioners interested in preventive medicine what is being done in the Old Country. Efforts of the kind described may appear to be slight, but they will not be wasted. The profession and the public in Australia have inquiring minds and would probably benefit from action taken along the lines adopted in Britain.

Current Comment.

CORTISONE AND THROMBOEMBOLISM.

THE complications which lie strewn across the path of cortisone therapy provide sufficient thought to deter the physician from the use of corticotrophin or of cortisone compounds unless they are strongly indicated in a particular case. These complications are a consequence of the profound alteration of circulating adrenal cortical hormones either by stimulation of the patient's cortex or by artificial hormone additions. Side effects include euphoria, virilism and a predisposition to infection by the tubercle bacillus. More serious complications are ulceration, bleeding and perforation of the gastro-intestinal tract, hypertension, water retention, and a striking aberration of electrolyte metabolism. The effects of cortisone may be disastrous in patients in whom there is already a mild, subclinical metabolic disturbance. Hypertension is increased, and several other abnormalities are exaggerated.

It is widely held that atherosclerosis is the terminal outcome of years of abnormalities of lipid metabolism. F. J. Stare[1] suggested that the serum lipid level might be maintained at high values in the presence of high dietary fat and cholesterol levels and in the absence of sufficient of certain of the amino acids. Thromboembolic phenomena are related to the presence of atherosclerosis and hence to the abnormally high blood lipid level. D. Adlersberg[1] showed that the most frequently encountered abnormalities in xanthomatous families are hypercholesterolæmia and coronary atherosclerosis. Genetic analysis supports the concept that the disturbance of cholesterol metabolism is inherited as a dominant trait. Studies of the families of younger patients with coronary atherosclerosis revealed familial high serum cholesterol levels, and Adlersberg found that the trait of hypercholesterolæmia was present in from 4% to 5% of patients admitted to a general hospital. From these studies it may be deduced that a fair proportion of patients who would benefit by the administration of cortisone-like compounds also suffer from hypercholesterolæmia, and hence are likely candidates for thromboembolic phenomena. Various papers have appeared which suggest that cortisone may produce a state of hypercoagulability of the blood in some patients, and this predisposes to thromboembolic phenomena. H. I. Russek, B. L. Zohman and A. S. Russek[2] expressed the view that this danger of cortisone therapy was relatively slight. In 86 patients with cardio-vascular or cerebro-vascular disease large doses of cortisone produced no complications. There was no indication for the concurrent use of cortisone and anticoagulants in such persons.

D. Adlersberg, J. Stricker and H. Himes[3] believe that thromboembolic phenomena do represent a serious complication during the use of cortisone or corticotrophin and that there is a special risk in those patients with a raised level of serum cholesterol. Of their patients, four, whose ages ranged from thirty-eight to fifty-two years, developed serious thromboembolic phenomena while they were receiving therapy by cortisone or corticotrophin. Examination revealed one common characteristic. Each had an abnormally high level of serum cholesterol. Families of three of the patients were examined, and the trait of hypercholesterolæmia was found in each. A fifth patient developed severe anginal attacks while under cortisone therapy. During the subsequent course anticoagulants were also given, and there was no angina. Later more cortisone was given but no anticoagulant, and the patient developed acute myocardial infarction. The serum cholesterol level was at about 500 milligrammes per 100 cubic centimetres of blood.

This series of patients of Adlersberg *et alii* cannot be taken as conclusive evidence that cortisone produces a particularly high rate of thromboembolic phenomena. There is no mention of the actual number of patients treated. It may be that the number of such patients with high serum cholesterol levels is such that some thromboembolic phenomena might have been expected regardless of the nature of therapy. However, it would seem that caution is advisable in the steroid therapy of patients with high serum cholesterol levels or with a familial tendency to atherosclerosis. The role of anticoagulants in cortisone-treated patients is not so clear. If, on the original classification of Russek, they are to be regarded as "good risk" patients, then the administration of anticoagulants would scarcely be justifiable.

ALCOHOLISM.

THE pursuit both of pleasure and of escape through the artificial aid of natural or synthetic chemical compounds is doomed to ultimate failure. If by means of any drug, man seeks to channel and to concentrate pleasure, to borrow future ecstasy, then he has failed in his duty to himself, he has rejected the realities of the world and has stepped, however little, out into the dark shadows of psychopathology. Of all fools the alcoholic is the greatest,

[1] *Am. Geriat. Soc.*, October, 1955.

[1] *Am. J. Med.*, November, 1951.
[2] *Am. Heart J.*, May, 1954.
[3] *J.A.M.A.*, December 31, 1955.

for he fools himself until the unreal image of the alcoholic dream flits away. Alcoholics are the untidy flotsam and jetsam of society. Under the influence of real or imagined stress they seek their escape, and under the influence of alcohol they cause mischief and misery in their homes. Chronic alcoholism is one of the greatest of menaces to modern civilization, and by its very existence is an accusation of failure against society itself.

Alcoholism is not a problem shared equally by mankind. It is the curse of the richest communities and of the poorest who would use it as a solace. It is no respecter of race or culture, and it is sometimes difficult to see by what the habit is induced and fostered. It is a problem said to be comparatively slight in Oriental races. It is a problem of appalling size in the United States of America. In France the position yearly grows worse, and drastic government controls are contemplated. In Sweden liquor rationing was recently abandoned with no grave signs that the excesses of past years will recur. In Britain the law is relatively lax, and the problem is comparatively small and is said to be decreasing.

The effects of chronic alcoholism on the human organism are widespread and well known. Most recently A. W. Williams[1] has suggested that there is no proof that alcohol is directly implicated in the production of peptic ulcers, and that the histological appearance of the stomach of alcoholics is frequently normal. Hepatic cirrhosis is brought about by the dietary deficiencies of the alcoholic. It may be that the fat alcoholic would in other circumstances over-eat and still be fat and atherosclerotic.

The treatment of chronic alcoholism is difficult, long and rewarded by frequent relapses. It is a problem likely to dismay the most eager psychiatrist, though, as J. F. J. Cade[2] pointed out, it is a problem which must be shared by the community. D. J. Feldman[3] suggests that in the light of our present knowledge total abstinence is the only possible aim of treatment. "Normal alcohol drinking" is impossible for such patients. Adrenal hypofunction may be rather common in alcoholics, but routine endocrine treatment has no significant effects in the general course of chronic alcoholism. There is no evidence that the condition arises from a specific metabolic need which could be treated by hypernutrition. Psychotherapy aims at convincing and supporting the patient in his belief that as he is powerless over alcohol he must live completely without it. The patient is supported and encouraged in this outlook by the organization known as Alcoholics Anonymous. This society induces a sense of group responsibility and recognizes that the alcoholic is never cured and must practise continual awareness of the effects of the first drink. Feldman states that "Antabuse" therapy has proved to be safe and that the antihistamines are effective antidotes in the event of reaction. Small maintenance doses are quite sufficient. As elimination of the drug takes from two to seven days, there is adequate time for second thoughts in patients who plan their drinking bouts. Creating a conditioned reflex of revulsion to alcohol by the use of emetine or apomorphine is of doubtful value. Psychoanalysis of the individual is useful only in selected cases. Group therapy is of much greater value, especially when associated with a sympathetic physician or the advice of a social worker or clergyman. Punishment as such has no place in treatment, but by threat of punishment the individual may be started in his rehabilitation, and it will bring together patients who would otherwise refuse to seek treatment. T. D. Rana[4] suggests that the alcoholic is emotionally immature and unstable and that his problems result, perhaps early, in tensions leading to relief by blotting out with alcohol. The narcotization of anxiety becomes habitual, and at the same time alcohol lowers still further the subject's power of inhibiting tensions. Like Feldman, Rana has found group psychotherapy to be the most valuable form of treatment. "Antabuse" treatment, he thinks, is not the solution to the problem of alcoholism, but may help to sustain total abstinence.

The difficulties in the treatment of chronic alcoholism are severe and challenging. The whole question of alcohol is a grievous social problem. Total prohibition is impracticable. For the majority of drinkers there is no problem. The introduction of young people to alcohol is a heavy responsibility, and only the passage of years reveals those whom the first drink leads to the social gutter of chronic alcoholism.

[1] *Brit. M. J.*, February 4, 1956.
[2] M. J. AUSTRALIA, March 3, 1956.
[3] *Ann. Int. Med.*, January, 1956.
[4] *Antiseptic*, February, 1956.

MALARIA IN EUROPE.

OF all the diseases suffered by mankind malaria is probably the cause of more reducible morbidity and early death than any other. The problem is primarily one of prevention by control and by the elimination of the anopheline mosquito. Particularly in the last decade medical science has moved with success to rescue whole areas from the plague of malaria. Perhaps the most notable example of control is that of Cyprus, where British action has virtually rid the whole island people of a serious scourge. The World Health Organization is intimately concerned with the problem of malaria, and it is not too much to hope that, providing the political situation does not deteriorate, malaria as a common disease will cease to exist within the lifetime of the average man of today. The problems which will then beset mankind as the chain of circumstance is removed from vast underprivileged populations are sufficiently difficult and do not concern us here.

Malaria is so frequently referred to as a disease of the tropics that it is regarded as a tropical disease. This supposition is untrue. Malaria exists wherever the anopheline mosquito can survive. Malaria was rampant in Great Britain until public hygiene and intensive agricultural development destroyed the breeding grounds. On the continent of Europe malaria is still a considerable problem in low-lying or in unenlightened areas. Holland is one of the most highly civilized, organized and affluent countries in Europe, and it comes as some surprise to realize that malaria is a considerable problem in the north of that country. It is interesting to note how the disease is dealt with in this area, and such a description has been given by H. Kraan.[1] The vector is the *Anopheles maculipennis*, which breeds from mid-April to mid-October and causes a malaria season from March to October with summer and autumnal peaks. The malaria type is *Plasmodium vivax*, and the largest number of notified cases occurred in 1948. Presumably because of the particular geography of the Province, the method of control of the mosquito was that of spraying with insecticides in dwellings in which the infected mosquito might be expected to lurk. Selective spraying of infected houses was considered to be adequate, though after the middle of August spraying had to be repeated every two weeks. Strip spraying and routine two-yearly spraying have long been practised in the Netherlands. It was found that the largest numbers of infected mosquitoes were in the houses of healthy parasite carriers. The point was then to detect the habitation of these carriers. General blood film examination of the entire community was impracticable. Detection was conducted by the examination of school children for splenomegaly and the blood film examination of members of such affected families. Later splenic enlargement was taken as the sole indication for the need of house spraying. The cooperation of the public in notifying malaria sufferers was sought, and many cases were later confirmed which had escaped notification by medical practitioners. Some infections were old and had originally been contracted in Indonesia or in other places. The infected dwellings were sprayed fortnightly with pyrethrum from 1941 onwards. However, the number of new cases continued to rise and the rate of splenic enlargement to increase in the houses which previously had not appeared to be infested, but in

[1] *Doc. Med. Geogr. Trop.*, December, 1955.

which there were undetected healthy carriers. Spraying with a 5% solution of DDT was commenced in 1947, and from this time no primary case occurred in treated houses, and in one year morbidity rates fell in one region from 51 to four per thousand of the inhabitants. The residual action of DDT also enabled earlier spraying to be carried out before the appearance of sporozoite-infected anophelines in the middle of August. Spraying was continued for three years in the house of each carrier. Preparations of DDT were made which did not contain kerosene and aroused no social objections. Trials of spraying all houses in a community were made in several areas with such success that the mosquito completely disappeared, even though its breeding grounds were not treated. However, this universal house spraying was too expensive for general use. In the various campaigns it was found that three to four litres of solution were required per household selected for spraying, and these covered between one-sixth and one-fifth of the houses in each community. The result of malarial control by selected spraying was such that in North Holland the incidence of the disease is now one of the lowest recorded, and in the whole of the Province only 200 houses still needed spraying by 1953.

EVALUATION OF SLEEP.

THE mechanism of natural sleep remains one of the mysteries of animal life. The lives of all complex terrestrial creatures are governed by the daily turning of the earth and the spreading of sunlight across its surface. There is a daily period of immobilization when only functions essential to life are active, and when the highest centres of sensory appreciation and protective calculation and, in man at least, consciousness are lost. The physiological changes which occur when the organism is asleep are little better understood. Anoxia, hypothermia, toxæmia and injury to central brain areas induce conditions of sleep-like nature; but investigation of normal patterns must be made in normal sleep, and the technical considerations of such investigations are considerable.

B. F. Hoffman, E. D. Suckling, C. McC. Brooks, E. H. Koenig, K. S. Coleman and J. H. Truemann[1] have made some progress in the qualitative evaluation of the physiological changes occurring in the human organism during sleep. Preliminary recordings were made on three conscious subjects over a period of five days. Readings of rectal temperatures and skin temperatures of the right great toe, anterior chest and interscapular regions were taken by means of thermocouples at two-minute intervals. Metabolic rate was determined at half-hourly intervals, and oral temperature and radial pulse rate were determined every five minutes. Correlation of the results obtained over periods of relaxation, meal times *et cetera* revealed that in two subjects there was a significant association between the metabolic rate and the toe, mouth and chest temperatures. This relationship was not found in the third subject, who had an elevated basal metabolic rate. Heart rate was significantly related to metabolic rate in two of the subjects. Rectal temperature did not relate to the other variables.

The second trial of procedures by Hoffman *et alii* concerned studies of the results obtained from six human volunteers during five nights a week for fifty to fifty-two nights per subject. Subjects slept in air-conditioned, sound-deadened rooms maintained at 72° F. Electroencephalographic recordings were made for one minute at intervals of seventeen and a half minutes. Similar electromyelographic recordings were made from the right biceps and the right gastrocnemius muscles. Bed movements were recorded by mattress bellows, and skin resistance was recorded between the palmar surfaces of the left and right hands. A loudspeaker system enabled alterations to be made to the various depths of unconsciousness by means of auditory stimulation. Results show that the depth of sleep can be related to changes in the electroencephalographic recording. Individuals tended to maintain characteristic levels of activity, and differences between individuals varied considerably. No relationship was found between the depth of sleep and the changes found by electromyelography. Skin resistance revealed a progressive change during the night in many instances, but could not be related to the quality of sleep. Heart rate fluctuated during sleep, but average volumes did not vary significantly with waking values. Body movements did occur mostly during periods of lightening unconsciousness and tended to follow the increase of electroencephalographic activity. The authors conclude from all these results that estimations of the depth of unconsciousness can best be made by periodic electroencephalographic records, that the heart rate is the best guide to general metabolism, and that the best way of detecting changes in metabolism is by the regular estimation of toe temperature.

[1] *J. Appl. Physiol.*, January, 1956.

MODERN PUBLIC HEALTH.

WHAT is "modern" public health? Those who qualified in medicine thirty or forty years ago are likely to think of public health as an affair of drains and stinks, with something about trying to control the spread of infectious diseases. A little was learned in those days about sanitary engineering, ventilation, purification of water, care in food handling, sanitation of houses, burial of the dead, abolition of filth, and the laws on health matters. Study had to do not so much with the man as with his environment. The practitioner's only definite impact on people came when he dealt with infectious diseases—vaccinating against smallpox, and isolating sufferers and contacts with the common infectious diseases.

Time makes many changes in language, and it is common for words to become altered in their meaning. For instance, the word "sanitary"—wholesome though its origin and infancy—has become cramped and even debased in meaning. When Chadwick wrote his report on the "sanitary condition" of the labouring population, he referred not merely to drains and cesspools but to the whole environment of the people. Yet, however much we may prefer "sanitary" in that wide sense, current usage must win. Only recently the council of the long-established Royal Sanitary Institute saw fit to adopt a new name—the Royal Society of Health.

Today the range of public health activity is wide, as wide as medicine itself. To Professor I. G. Davies, "modern public health" means much more than privies, drains and stinks. He is qualified to speak with authority on the subject, for he is Professor of Public Health in the University of Leeds and medical officer of health and school medical officer for the City of Leeds. He is the author of an important volume[1] which has little reference to drains or water supply or waste disposal or camp hygiene or cowyards or slaughterhouses. He mentions that those things comprised the concept of public health in the nineteenth century. What Professor Davies describes as modern public health has evolved in the last twenty or thirty years. His interesting and useful survey of the organization and functions of public health at the present day is based on the developments in Britain. Modern public health has made a shift to the personal; health environment is now taken for granted, and doctors are asked to focus their attention on the people moving in it. Social work has become a major interest to the health administrator; in Britain public health nurses are now designated health visitors, and most of their work brings them to people's homes. Problem families call for much attention. Psychiatric social workers take an important place in the health departments of many American cities. Thus the new work has to do with people rather than germs and inanimate things. The terms "human ecology" and the

[1] "Modern Public Health for Medical Students", by I. G. Davies, M.D., F.R.C.P., D.P.H.; 1955. London: Edward Arnold (Publishers), Limited. 8¾" × 5¼", pp. 494. Price: 30s.

"epidemiology of health" have come into current use, and the range of study they cover goes far beyond the germ-caused outbreaks of illness. Health clinics, nutrition, preventive psychiatry, medical care, rehabilitation, and health education—those are the fields of interest in public health today. But although the basis of modern public health is clinical medicine, we still cannot afford to neglect the old-time principles of sanitation.

Professor Davies goes into some detail about genetics and the relation of heredity to public health. That is an interesting study, but in few countries can the principles be applied vigorously in health administration. We can do little more than speculate.

Doctors in this country will not interest themselves much in the summary given by Professor Davies about the *National Health Service Acts* of Great Britain; but they would do well to study his discussion on diseases of special social importance. Here are covered such important subjects as cancer, cardio-vascular disease, rheumatism, epilepsy, diabetes and peptic ulcer. This is "modern" public health. So are studies on communicable diseases, the welfare of the aged and of handicapped persons, occupational health, maternal health and infant welfare, subjects which have been dealt with from time to time in the editorial columns of this journal. They are handled in a clear and concise fashion by Professor Davies.

We have given emphasis to this book because it must be accepted that the old-time public health work has now passed out of the doctor's hands. Sanitary engineers, town planners, health inspectors and health visitors all do that fundamental work in early public health. Medical men have done their part in developing those ancillary branches and in fostering legislation on environmental hygiene. Today the doctor in public health can attend to matters closer to his training and clinical interests. Now that public health rests firmly on a basis of clinical medicine, the good public health doctor must first be a sound clinician. Several text-books on public health in recent years have propounded that theme. The book of Professor Davies will help medical men in the understanding and performance of public health in present-day community life.

SYDENHAM'S CHOREA AND THE STREPTOCOCCUS.

THE characteristic signs of chorea in childhood were described almost fully by Thomas Sydenham towards the end of the seventeenth century. Chorea is a disease which has fortunately tended to lessen in frequency with the improvements in social conditions. The child with chorea is an unhappy creature. His purposeless twitchings, his clumsiness, his perpetual restlessness and emotional lability and his slurred speech in the absence of any obvious illness have led to family intolerance and neglect. It was only in modern times that chorea or Saint Vitus dance fell under suspicion as a real disease. The frequent findings of nodules resembling those of rheumatism, the appearance of *erythema marginatum* and the association with subsequent or succeeding acute rheumatism led to the conclusion that the chorea was in some way related to the rheumatic process. B. M. Kagan and B. Mirman[1] suggested that there were two types of Sydenham's chorea. In one, associated with an increase in the sedimentation rate, the disease was a manifestation of active rheumatic fever. The other group was non-rheumatic in origin and was not associated with an increased susceptibility to acute rheumatism. The ætiology of this second group was unknown though it seemed to be related to psychogenic factors. W. Gaisford and R. Lightwood[2] expressed the now prevalent view that chorea was always a manifestation of the rheumatic state and that in almost half the cases it was associated with typical rheumatic carditis leading to mitral stenosis. The presence of a raised erythrocyte sedimentation rate suggests active carditis, and there is frequently a seven to ten day history of group A streptococcal infection. There is often evidence of past rheumatism and changes in electrocardiographic findings. Gaisford and Lightwood suggested that the ætiology of chorea was probably associated with antigen-antibody reaction following the streptococcal infection, though at that time there was no direct evidence for this view. The treatment of chorea now runs a similar course to that of acute rheumatism, of which it is now almost certainly a less frequent form.

The actual relationship of chorea to the group A streptococci has been studied by A. Taranta and G. H. Stollerman.[1] In 10 children whose only signs were those of pure chorea, within five months of the onset high titres of antistreptolysin were found in eight. Of 30 other children in whom chorea was one of the manifestations with cardiac involvement, 22 had high titres within one month of the onset; of 11 children studied later in the disease five had a similar high antistreptolysin titre. From other data it appears that the incidence of such a high titre is almost the same in the first month of chorea as in the second month of acute rheumatism. There was no significant statistical difference between the antistreptolysin titres on the cerebro-spinal fluid of the choreiform and the rheumatic patients. A study of the community seasonal incidence of chorea shows that it tends to lag about two months behind that of acute rheumatism. In fact chorea tends to be a late manifestation of acute rheumatism when these changes occur. The latent period between streptococcal infection and chorea is longer than that of acute rheumatism and may be up to seven months, though chorea does not occur longer than nine months after the elimination of the streptococci. The "pure" chorea may in fact follow some considerable time after subclinical rheumatism. Chorea, like *erythema marginatum*, is a late rheumatic sequela, although it may appear when the actual titre of antistreptolysin is falling.

From these and other studies it has been concluded that chorea not only has a common origin with acute rheumatism, but is in fact a late manifestation of the rheumatic process. By implication, the early recognition of the choreiform state becomes even more important, and the inclusion of these children under the antistreptococcal shelter of continuous therapeutics must be considered. The intricate tangle of the web of the collagen diseases is the Gordian knot of medicine. Those who haplessly dance before the image of Saint Vitus are still more victims of a common mystery yet unsolved.

[1] *J. Pediat.*, September, 1947.
[2] "Pædiatrics", Volume I, Butterworth, London, 1953.

SERUM CHOLINESTERASE IN DIABETICS.

STUDIES of the incidence of various abnormalities have tended to relate together such conditions as myocardial infarction and *diabetes mellitus*. In conditions conducive to atherosclerotic changes in blood vessels there may be a long-standing dyscrasia of lipid metabolism. How far such aberrations of the physiological fate of endogenous fat can be related to the diabetic state remains uncertain, but it has been suggested that in diabetics the plasma cholinesterase level is usually raised. R. H. S. Thompson and J. R. Trounce[2] have determined the serum cholinesterase levels in a series of diabetic patients grouped according to the degree of obesity and the adequacy of insulin control. A similar series was conducted upon both obese and normally healthy subjects. The results reveal that in the overweight diabetic patients there was a high level of serum cholinesterase unrelated to the severity of the diabetes. The average level in obese but otherwise healthy subjects was also rather high. There was no significant difference between the levels in the diabetic and healthy subjects of normal weight. These results suggest that the influence of the diabetic state upon the level of serum cholinesterase is, if anything, slight. The main factor is the obesity.

[1] *Am. J. Med.*, February, 1956.
[2] *Lancet*, May 12, 1956.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Comparative Studies of Intestinal Amœbæ.

C. T. PAN AND Q. M. GEIMAN (*Am. J. Hyg.*, July, 1955) have begun a comparative study of intestinal amœbæ by observing the distribution and cyclic changes of nucleic acids in *Entamœba histolytica* and *Entamœba coli*. Their strain of *E. histolytica* was pathogenic for hamsters, and it was maintained in Locke-egg-rice starch medium with multiple bacterial flora. The same method was used for *E. coli*. The amœbæ were harvested on half coverslips after varying intervals of growth. They were fixed, hydrolysed for seven minutes in normal hydrochloric acid, and stained by modifications of the Feulgen technique for desoxyribonucleic acid. The containers were kept in the dark. Ribonucleic acid was demonstrated after fixation in Carnoy's fluid, and the slides were treated with ribonuclease before being stained. Results showed that trophozoites of *E. histolytica* did not always give a Feulgen reaction in the nucleus; when it did so the nucleus was in metaphase or near to that stage; as division ended, the desoxyribonucleic acid disappeared. In the cysts the Feulgen reaction for desoxyribonucleic acid appeared only in the early binucleated forms. *E. coli*, on the other hand, constantly showed desoxyribonucleic acid in 95% of the trophozoites after eight to twelve minutes' hydrolysis, and during divisions the desoxyribonucleic acid granules could be seen moving and reassembling in the position of the daughter nuclei. Ribonucleic acid was present in both species, and in the cysts it was in the chromatoid bodies.

Immune Response to Foreign Protein.

J. C. ROBERTS AND F. J. DIXON (*J. Exper. Med.*, October, 1955) used the transfer of lymph node cells in the study of the immune response to foreign proteins. Rabbits were immunized with bovine γ globulin or serum albumin by the subcutaneous or the intravenous route, antibody response was tested a week later, and one week later the animals were sacrificed and lymph nodes were excised and treated to produce cell suspensions. Test rabbits were treated with X rays to inhibit primary immune response, and then each was given an inoculation of antibody containing cells either by the subcutaneous or by the intramuscular route, and then varying amounts of I^{131} labelled antigen were inoculated intravenously. Determinations of circulating antigen and of the amounts of antibody in the injected cells were then made. The calculations of the number of cells inoculated and the antibody which they carried appeared to represent approximately two-thirds of the wet weight of those cells. Normal cells did not elicit any evidence of antibody when used in control animals, nor did incubation *in vitro* of normal cells and labelled antigen reveal any evidence of antibody production.

Kaposi's Varicelliform Eruption.

R. C. B. PUGH, J. A. DUDGEON AND M. BODIAN (*J. Path. & Bact.*, January-April, 1955) describe a fatal case of Kaposi's varicelliform eruption with typical and atypical visceral necrosis. The infant had had skin eczema for some time and was admitted to hospital, where examination of vesicle fluid showed multinucleate epithelial cells often associated with Kaposi's varicelliform eruption. Two nurses and two other children developed vesicular skin eruptions, and herpes virus was identified in suckling mice and rabbits, guinea-pigs and adult mice. The histological appearances are described in detail, including some unusual necrotic lesions from which virus was not obtained *post mortem*.

Chemical Basis of Virulence of Bacillus Anthracis.

H. SMITH, J. KEPPIE AND P. HARRIS-SMITH (*Brit. J. Exper. Path.*, June, 1955) have continued their study of the chemical basis of virulence by determining the role of the terminal bacteriæmia in the death of guinea-pigs from anthrax. They used a dose of 2×10^9 spores given by the intradermal route, which produced death of the animal from three to five days later with a bacterial count in the blood of 1×10^9 chains of the organism per millilitre. The infections could be controlled by doses of streptomycin, which rapidly cleared the blood of organisms, provided it was given when the blood had less than 3×10^6 organisms per millilitre. This degree of multiplication of the organisms was the factor which determined the death of the animal within the following three days. Over half the organisms at death were concentrated in the spleen, and the failure of the spleen to clear the blood of organisms was followed by rapid death.

The next paper in this series (*ibidem*) discusses the death from secondary shock exhibited by the animals; and evidence of the fall in blood volume and pressure disturbances of electrolyte balance and metabolic function leading to loss of muscular tone is set out. Histopathological studies also showed the changes associated with renal failure.

H. SMITH, J. KEPPIE AND J. L. STANLEY (*Brit. J. Exper. Path.*, October, 1955) have continued their investigation of the chemical basis of the virulence of *Bacillus anthracis* and the specific toxin produced *in vivo*. The œdema-producing factor was tested in guinea-pigs dying of the infection, and it was found present in greatest amounts in the plasma and in the thoracic exudate; the plasma activity was greatest when the number of organisms in the blood was greatest, and the reaction was specific. When the plasma was dried and stored, its activity was preserved, while that of the liquid plasma progressively decreased. Plasma activity was lost on heating to 60° C. for thirty minutes. Activity was altered by any change of pH from 7 to 8 and almost absent at pH 5 or 10. Fractionation of the toxic plasma by various means always resulted in a great loss of activity, although some tissue-damaging factor could always be found in the fraction which contained the protective antigen. The toxin appeared to be lethal in mice and guinea-pigs in proportion to its œdema-producing ability, and to be in no way connected with the capsular substance. The death of the guinea-pig after an injection of anthrax toxin was produced by secondary shock.

Influenza Virus in Immune Environments.

T. P. MAGILL (*J. Exper. Med.*, September, 1955) planned to study the propagation of influenza virus in immune environments. He immunized groups of mice with formalin-killed virus injected into the peritoneum, using increasing doses for the purpose in different groups of mice. He then passaged by intranasal culture the same virus which was used to produce immunity and studied the strains recovered from the lungs of some of the test mice in each group, taking especial care that the variants obtained were not contaminated during manipulation. Hæmagglutination tests with selected antisera showed that there was a resemblance to strains isolated in 1951 rather than the parent strain. The mice immunized with high titre virus still showed high titre of virus in the lung lesions; so the process was continued, and further variants emerged. The point seemed to be established that under the challenge of an immune environment, the virus could survive; and once survival was achieved, a genetic modification of the virus had been induced, which maintained itself in the face of increased immune environments.

Proteolytic Activities of Corynebacteria.

A. J. LUZZIO AND J. O. HARRIS (*J. Infect. Dis.*, September-October, 1955) have studied the proteolytic activities of certain members of the genus *Corynebacterium*. They used poured plates containing seven different protein substrates; these were egg albumen, α globulin, casein lactalbumin, lactoglobulin, litmus milk and gelatin. Thirteen species of corynebacteria were tested. *Corynebacterium pyogenes* and *Corynebacterium helvolum* were active against all substrates, but the great majority of strains were inactive. The authors note that all substrates were subjected to greater or lesser degrees of heat; therefore the proteins were partially denatured, and the results must be examined with that fact in mind.

Experimental Meningitis.

M. HAMBURGER, K. L. CLARK, J. P. BIEHL AND L. P. JERVEY (*J. Infect. Dis.*, July-August, 1955) initiated a series of studies of experimental meningitis in Rhesus monkeys by observing the pathogenic effect of various bacteria recovered from human cases. Difficulties were encountered in preparing bacterial inocula, and the most satisfactory method proved

to be the grinding up of jellied agar containing saline dilutions of bacteria, thinned with beef broth, and the inoculation of one millilitre amounts into the *cisterna magna*, the animal being anæsthetized with "Pentothal". An attempt was made to assess the amount of exudate present, and pneumonocci and *Pseudomonas æruginosa* produced the greatest amount, while those animals inoculated with *Hæmophilus influenzæ B* or *Streptococcus salivarius* failed to develop meningitis. When *Pseudomonas æruginosa* was suspended in agar, predetermined doses led to a reproducible meningitis, and the cerebro-spinal fluid contained polymorphonuclear leucocytes, fibrin and extracellular desoxyribonucleic acid; this technique is suggested as a model for the study of therapeutic measures in meningitis.

HYGIENE.

Benzene Content of Petroleum Solvents.

H. E. ELKINS AND L. D. PAGNOTTO (*Arch. Indust. Health*, January, 1956) describe methods of analysis of petroleum fractions for benzene. The benzene concentrations of eight samples of naphtha solvents ranged from 0·6% to nearly 7%. The authors consider that the use of solvents containing around and above 5% of benzene in processes such as fabric spreading might lead to excessive exposure to benzene.

Absence of Pneumonokoniosis Among Workers in Pure Limestone.

S. B. DAVIS AND G. NAGELSCHMIDT (*Brit. J. Indust. Med.*, January, 1956) report the results of an investigation to determine the effect of pure limestone dust in the lungs. Clinical and X-ray examinations were carried out on the men, who had worked with limestone for periods varying from twelve to thirty-five years. The limestone had an average content of 97% of calcium carbonate and less than 1% of quartz. Dust counts at the time of the investigations showed many of the order of 3000 particles per cubic centimetre of a size between 0·5 and 5 microns. It is likely that dust concentrations would have been higher in previous years. No evidence of pneumonokoniosis or bronchitis was found in any of the men investigated.

Fibrogenic Activity of Different Forms of Silica.

S. H. ZAIDI, E. J. KING, C. V. HARRISON AND G. NAGELSCHMIDT (*Arch. Indust. Health*, February, 1956) describe in two articles the results of two experiments. In the first, different forms of free silica, that is, fused silica, quartz, cristobalite and tridymite, of high purity and almost identical silica solubility, were injected intravenously into the tails of mice to study the pathological changes produced in the liver. Tridymite was the most rapid in producing acellular collagenous silicotic nodules, cristobalite was next, quartz was the third, and fused silica was the least fibrogenic. Silica particles were found only initially in the spleen, and very little fibrosis was produced there. No fibrosis was observed in any other tissue. Since these dusts have the same solubilities, the authors suggest that there must be some other factor, possibly related to the character of the particle surface, responsible for the different pathogenicities. In the second experiment, flint dusts of several sizes ranging from less than 0·5 μ to 4·0 μ were given to rats by injection into the trachea. The amounts of the different size fractions were chosen to yield equal surface areas of the particles contained. The optimum size of flint particles to yield maximum fibrosis in the lungs of rats was found to be between 1 μ and 2 μ in diameter. Samples of flint dust of different sizes in fractions of less than 0·2 μ to 4 μ were administered to mice by the intravenous route. The amounts of the different size fractions used had equal areas of particle surface. The optimum fibrogenic size of flint particles in the livers of mice lay between 0·2 μ and 2 μ in diameter, a wider range than that found in the lungs of rats. Particles of flint smaller than this, and larger, were less fibrogenic. The results, according to the authors, support the conception that fibrogenesis is closely related to the silica surface, but that there is also a range of maximum fibrogenic sizes.

School Health Ventilation.

C. P. YAGLOU (*Am. J. Pub. Health*, February, 1956) considers that the basic requirements for heating and ventilating schools are a comfortable temperature with moderate humidity and sufficient outside air to avoid accumulation of unpleasant odours. Fresh air requirements are discussed. Bacterial content of air has been shown to have no relation to the spread of acute infectious diseases in an occupied room. The minimum amount of fresh air for odour control in schools is ten cubic feet per minute per pupil. It has also been shown that the incidence of upper respiratory tract infections is related to the number of persons housed in a room and not to the floor area or cubic space allowed per person. Natural and artificial methods of obtaining fresh air without setting up draughts or steep temperature gradients are described. The author considers that the ventilation regulations that apply to New York State are rational and up to date. These regulations require that heating systems be designed and guaranteed to maintain certain specified temperatures in different sections of the school, with a vertical gradient not exceeding 5° F. from the floor to the 60-inch level. The ventilating system must provide a minimum air change of 10 cubic feet per minute per pupil to remove odours without producing draughts. The air movement in zones of occupancy must not exceed 25 feet per minute. For effective thermal operation in mild weather a design factor of 15 cubic feet per minute is recommended. Where extensive use of a school building is anticipated in hot summer weather, the department may require installation of an air-conditioning system, or the use of electric fans. Special ventilation systems are required where toxic substances or strong odours are produced. Authority to approve plans and specifications for school buildings is vested in the Commissioner of Education.

The Health Officer in the Control of Viral Diseases.

J. E. GORDON (*Am. J. Pub. Health*, February, 1956) discusses the health officer's part in the control of virus diseases. He refers to "the stream of shining discoveries that continues to come off the scientific assembly line" and suggests that the role of the health officer is to follow these discoveries and relate them to the diagnosis, epidemiology, management and prevention of communicable diseases. The information should then be made available to the practising physician. Local and central authority health officers have different roles in the control of disease. All require the assistance of the laboratory. The local officer is in intimate contact with the patient and his associates, and has opportunities to study the epidemiology. The health officer of the central authority is responsible for expert advice, laboratory facilities and evaluation of control procedures. In the reporting of disease, the author refers to the value of reporting epidemics in addition to individual cases. In discussing control the author divides diseases into groups according to the degree of effective control at present available. Rubella and chicken-pox belong to a group for which control is not effective. The common cold belongs to a group where reasonable control procedures produce no material result. Active and passive immunization are effective for another group which includes smallpox, infective hepatitis, measles and poliomyelitis. The control of a group of diseases of presumed viral origin which includes infectious mononucleosis is still a matter of research. Reference is then made to the use of special methods of control for a number of virus diseases including smallpox, infective hepatitis and poliomyelitis.

Ground Disposal of Radioactive Wastes.

R. J. MORTON AND E. G. STRUXNESS (*Am. J. Pub. Health*, February, 1956) draw attention to the problems associated with the disposal of unused by-products of nuclear energy operations. They discuss the future of the inevitable atomic energy development and conclude that in the United States alone 400 nuclear power plants will be required by the year 2000. Radioactive wastes are now stored in tanks, and some wastes require storage for two hundred years before they are safe to release. Storage therefore has limitations. Processes to remove the more hazardous radioactive wastes are being investigated. Disposal of radioactive waste in the ocean is being investigated for its effect on marine life and marine food supplies. Other problems relating to dispersal by ocean currents are being reviewed. Disposal of radioactive wastes into pits in the ground for complete retention or for partial retention and seepage is discussed. The latter will depend on adequate adsorption and the ion exchange capacity of the soil. These will need to be determined.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the Western Australian Branch of the British Medical Association was held at Tintein Lodge, Perth, on March 17, 1956, DR. B. W. BUTTSWORTH, the President, in the chair.

RETIRING PRESIDENT'S ADDRESS.

Dr. Buttsworth delivered his Retiring President's address (see page 949).

ANNUAL REPORT OF COUNCIL.

The annual report of the Council for the year ending March 17, 1956, was received and adopted. The report is as follows.

The President and members of the Council of the British Medical Association (Western Australian Branch) have much pleasure in presenting the fifty-seventh annual report of the Branch for the year ending March 17, 1956.

Membership.

The membership of the Branch has increased during the twelve months ended December 31, 1955, by a net amount of 49, as follows:

Membership at December 31, 1954		529
Losses—		
Transferred to other Branches	11	
Resignations	5	
Deceased	4	
	—	20
		509
Gains—		
Transferred from other Branches	43	
Elections	26	
	—	69
Membership at December 31, 1955		578

Congratulations of the Branch are offered to Dr. J. J. Holland, who completed fifty years' membership of the Association on December 31, 1955.

Deceased.

With deep regret we record the deaths of the following members, and former members, which occurred during the year: W. E. Caldwell, F. L. Gill, B. J. Hallion, D. N. Kekwick, M. J. Morris, L. A. Wilson and W. A. Young. The sincere sympathy of the Branch is extended to the families of these late members.

Meetings.

In addition to the annual meeting, seven general meetings of the Branch were held. The subjects of these meetings were as follows:

April: "The Role of the General Practitioner, the Gynæcologist and the Radiologist in the Management of Carcinoma of the Cervix", Dr. Stenning.

May: "Town Planning and Health", Professor A. E. Stephenson.

June: "Recent Advances in Orthopædics", Orthopædic Services of the Royal Perth Hospital.

August: "A Symposium on Extrapulmonary Tuberculosis", Royal Perth Hospital.

September: "Science and the Law, with Special Reference to the Expert Witness", Sir Wilfred Fullaghar.

October: Clinical evening, Fremantle Public Hospital.

November: "The Workers' Compensation Act", Dr. H. L. Cook, Dr. H. M. Hill and Mr. G. D. Wright.

Council Meetings.

Twelve meetings of the Branch Council were held. The record of attendance is as follows:

B. W. Buttsworth (President)	11
C. W. Anderson (Honorary Secretary and Federal Councillor)	12
A. R. Bean (Councillor)	12
B. O. Bladen (Chairman, Contract Practice Committee)	7
F. W. Carter (Permanent Vice-President)	—
D. M. Clement (Assistant Honorary Secretary and Federal Councillor)	12
H. L. Cook (Vice-President)	12
D. E. Copping (Federal Councillor)	12
S. E. Craig (Councillor)	3
W. S. Davidson (Deputy Commissioner of Public Health) (deputizing for Dr. L. I. Henzell)	3
A. L. Dawkins (Chairman of Convocation)	11
D. W. Fleming (Councillor)	12
L. I. Henzell (Commissioner of Public Health)	7
A. Johnson (Commonwealth Director of Health)	3
D. D. Keall (Honorary Treasurer)	11
H. K. Pawsey (Councillor)	8
J. H. Stubbe (Past President)	3
A. B. Wilson (Councillor)	9

Office-Bearers and Councillors.

The following members have been elected as office-bearers for 1956:

President: Dr. H. L. Cook.

Vice-President: Dr. A. A. Merritt.

Honorary Secretary: Dr. C. W. Anderson.

Honorary Treasurer: Dr. D. D. Keall.

Honorary Assistant Secretary: Dr. R. L. Leedman.

Chairman of Convocation: Dr. A. L. Dawkins.

The permanent Vice-President of the Branch is Dr. F. W. Carter, who was elected to that position by resolution at the general meeting of June 17, 1953.

Federal Councillors elected for 1956: Dr. C. W. Anderson and Dr. D. M. Clement.

The following members have been elected as the five ordinary members of Council for 1956: Dr. B. O. Bladen, Dr. A. R. Bean, Dr. D. W. Fleming, Dr. M. Connaughton, Dr. R. S. W. Thomas.

At this juncture it is desired to express on behalf of the Association the warmest appreciation of the work done by the retiring councillors.

Federal Council.

Dr. Colin Anderson and Dr. Donald Copping, who on his resignation was replaced by Dr. Dixie Clement, were the representatives of the Branch on the Federal Council. Dr. B. O. Bladen acted as proxy for Dr. Clement at the February, 1956, meeting. Branch Council is deeply appreciative of the work performed by these members on behalf of the Branch.

The Federal Council at its meeting on February 27, 1956, recorded its appreciation of the valuable services rendered to the Federal Council and to the profession in Australia by Dr. Copping whilst a member of the Federal Council.

Library.

The library was moved to its new quarters in the Royal Perth Hospital during 1955. The general activities of the library will be the subject of a separate report.

Representation.

The Branch was represented at various meetings and conferences during the past year as follows:

Council of the British Medical Association: Dr. Miles Formby.

Federal Council of the British Medical Association in Australia: Dr. C. W. Anderson, Dr. D. M. Clement, Dr. D. E. Copping.

University Advisory Board in Medicine: Dr. H. H. Stewart.

Australasian Medical Publishing Company, Limited: Dr. F. W. Carter.

The Medical Journal of Australia: Dr. R. L. Leedman.

State Health Council: Dr. D. M. Clement, Dr. H. L. Cook, Dr. A. Gild, Dr. J. A. Love, Dr. H. K. Pawsey, Dr. J. R. H. Watson, Dr. A. B. Webster, Dr. M. F. Williams.

Nurses' Registration Board: Dr. L. E. LeSouef, Dr. R. H. Nattrass.

Dental Board: Dr. I. O. Thorburn.

College of Dental Science: Dr. H. E. H. Ferguson.

Workers' Compensation Act Underwriters' Joint Committee: Dr. H. M. Hill, Dr. G. B. Maitland, Dr. N. H. Robinson.

St. John Ambulance Association: Dr. A. L. Dawkins.

Optometrists' Registration Board: Dr. D. D. Paton.

Sex Education (Parents and Citizens' Association): Dr. I. O. Thorburn.

Protection of Practices: Dr. F. W. Carter, Dr. L. A. Hayward, Dr. H. H. Stewart.

Medical Services Committee of Inquiry: Dr. C. W. Anderson, Dr. F. W. Carter, Dr. H. L. Cook, Dr. H. J. Gray.

Medical School Appeal Committee: Dr. B. W. Buttsworth.

Health Education Council: Dr. D. E. Copping.

Anti-Cancer Council of Western Australia: Dr. J. G. Hislop.

Public Health.

We wish to take this opportunity of thanking Dr. Linley Henzell, Commissioner of Public Health, Dr. W. S. Davidson, Deputy Commissioner of Public Health, and Dr. A. Johnson, Commonwealth Director of Health, for their valuable cooperation and assistance during the year.

Australasian Medical Publishing Company, Limited.

Dr. F. W. Carter has continued with zeal to act as the nominated director of the Company for this State and as usual has kept the Branch Council fully informed with most comprehensive reports of directors' meetings. Owing to ill health, Dr. Carter was unable to attend the February, 1956, meeting, and Dr. C. W. Anderson attended as his proxy.

Contract Practice.

Again during 1955 this committee has had to do considerably less work than heretofore, but such as was necessary has been carried out with customary efficiency.

Post-Graduate.

This committee under the very capable chairmanship of Dr. Dixie Clement has had another very successful year, and its activities are the subject of a separate report presented by its honorary secretary, Dr. Adrian Lamb.

Social.

Medico-Legal Golf.—The annual golf match between the Law Society and this Association was held at Royal Perth Golf Club on October 12, 1955, and, despite the small number of medicos present, was a most successful day. The B.M.A.C. Trophy was won by Mr. Justice Jackson, the foursome trophy being won by Messrs. A. G. Smith and A. Bateman. It is to be hoped that more of our members will be able to be present next year. A permanent date has been set for this annual fixture—namely, the second Wednesday in October.

Convocation.

Two meetings of convocation were held during the year, and the main items for discussion were of a medico-political nature.

Council of Social Agencies.

The Federal Council has accepted foundation membership on the Australian Council of Social Welfare, and the Branch has assisted a proposal to form a Council of Social Agencies in Western Australia by providing the secretarial work involved in arranging a meeting of voluntary social agencies.

British Medical Agency Company.

British Medical Agency Company completed another successful year and again increased its contribution to the income of the Branch. The support from members could be greater, and all members are requested to use the facilities of British Medical Agency Company to their own and the Association's advantage.

Staff.

The Council wishes to express to the office and library staffs its sincere thanks for their cooperation during the year.

Induction of President.

Dr. Buttsworth introduced the President for 1956, Dr. Leigh Cook, and vacated the chair in his favour. Dr. Cook thanked the members for his election.

Medical Benevolent Association of Western Australia.

Dr. J. L. Day presented the annual report of the Medical Benevolent Association of Western Australia, which was adopted. The report is as follows.

The committee of the above Association has pleasure in presenting a statement indicating the receipts and disposal of funds for the year ended December 31, 1955.

Income was derived from subscriptions through the British Medical Association (£487 4s.) and from interest on Commonwealth loans (£54).

Expenditure was in the nature of benevolent contributions to widows and doctors in necessitous circumstances and totalled £871 5s. 6d.

During the year Mrs. Elliott, who had been a beneficiary for very many years, died.

Invested funds remain the same at £1700, Commonwealth Treasury bonds, which together with bank balance of £810 19s. 3d. total £2510 19s. 3d.

The committee expressed their sincere appreciation of the continued support of your Association.

Report of the Library Committee.

The report of the Library Committee was presented by Dr. E. Beech and adopted. The report is as follows.

We beg to submit to you the report of the committee on the British Medical Association Library for the twelve months ended March 12, 1956.

The committee met on 12 occasions during the year, and a report on library matters was submitted to Council each month.

A meeting was held between representatives of the British Medical Association, University, Library Committee and Royal Perth Hospital to formulate a broad general basis of agreement as to the conditions under which the library would be amalgamated with the University. Although this has not yet been finally ratified by Council and the University, members may be sure that there will be no loss to their library privileges enjoyed by them previously. We expect to keep up the same service to both town and country members. For instance, the library key can still be obtained from the Casualty Department, Royal Perth Hospital, after hours; the "In" and "Out" box, which was outside the old library, has been placed outside the back door of the library to enable members to deposit and pick up books after hours.

All members must now be aware that in October of last year the British Medical Association Library moved from the quarters it had occupied since 1949 to new and more spacious premises in the reconstructed and redecorated old out-patient department of the Royal Perth Hospital, to form the nucleus of what is to become the Central Medical Library of Western Australia when the Medical School comes into being next year.

The library has been established by the combination of the British Medical Association, University and Royal Perth Hospital libraries, the British Medical Association's initial contribution being the donation of all its present stock of books and journals. Although the furnishings of the new library are not yet complete, those members who visited it must, I am sure, have been delighted by what promise to be ideal surroundings in which to browse or study.

The change-over has, of course, precipitated a certain amount of confusion while the stock is resorted and placed once more within easy access on the shelves, and members have perforce been subjected to some inconvenience in obtaining the reference they wanted. However, within the last couple of weeks rapid strides have been taken in the reorganizing process, and things should be back to normal very shortly.

Members will be pleased to know that the plaque erected to the memory of Cyril Bryan which formerly hung in the old library will be rehung in the new, with an added inscription to suit its new location. It would be fitting also, we thought, if some form of commemoration to Harry Lucraft, who did so much to make the British Medical Association Library what it is, could be included in the new library. To this end we established last year, with Council's approval, a Harry Lucraft Memorial Fund, which reached the total of £22 1s. last March.

That the library is well used by its members is evidenced by the fact that approximately 3250 requests for information were received by the librarian, both personally and by letter and telephone. Six hundred and fifteen books were borrowed as well as 2868 journals. Country members also seem to be becoming increasingly aware of the assistance the library can be to them, and a large number of books, journals and photostats were sent to places as far apart as Geraldton and Denmark.

BRITISH MEDICAL ASSOCIATION (WESTERN AUSTRALIAN BRANCH).

Statement of Income and Expenditure during Twelve Months Ended December 31, 1955.

INCOME.

	£ s. d.	£ s. d.
By Members' Subscriptions		7248 3 0
„ Interest—		
(a) Commonwealth Bonds	51 10 3	
(b) Australasian Medical Publishing Company, Limited—		
(i) Debentures "A"-"D"	17 5 0	
(ii) Debentures "E"	53 9 1	
		122 4 4
„ British Medical Agency Company Revenue Account		828 15 11
		£8199 3 3

EXPENDITURE.

	£ s. d.	£ s. d.	£ s. d.
To Subscriptions to—			
(a) Members' Journals:			
(i) Australasian Medical Publishing Company, Limited	550 10 0		
Less Series "E" Debentures	274 17 6		
		275 12 6	
(ii) British Medical Association, London		866 8 9	
			1142 1 3
(b) Federal Council, Sydney			555 9 0
(c) Medical Benevolent Association			505 11 6
(d) Flying Doctor Service			1 1 0
(e) Historical Society of Western Australia			12 6
			2204 15 3
„ Cost of Administration—			
Advertising		13 19 0	
Audit Expenses		31 10 0	
Depreciation		51 13 6	
Duplicating		82 15 7	
General Expenses		64 5 5	
Legal Expenses		31 12 0	
Library Expenses		314 15 0	
Medico-Political Expenses		57 4 5	
Postages and Petty Cash		339 14 11	
Printing		34 11 6	
Rent		272 4 6	
Salaries—			
Secretary	1083 10 0		
Typists	757 3 8		
Librarians	986 18 0		
Clerical Assistant	134 5 7		
		2961 17 3	
Stationery		68 7 9	
Superannuation — Secretary		225 0 7	
Telephone		103 19 1	
			4653 10 6
			6858 5 9
„ Excess of Income over Expenditure			1340 17 6
			£8199 3 3

Sixty-three new books were added to the library, 27 of which were donated by THE MEDICAL JOURNAL OF AUSTRALIA. The Clinical Research Unit added 19 books to its section, all of which are readily available to members. Several large packing-cases of journals which we needed to complete our holdings have arrived during the year, sent to us as part of a world-wide library cooperative system of supplying missing numbers of journals to any library in need. We also were able to do our bit by supplying such libraries as the University of Liverpool, University of Birmingham, the Royal College of Physicians, London, Saint Mary's Hospital Medical School Library and the University College of Uganda, East Africa, with material they needed.

We would like to thank all those members and associations who have donated books and journals to the library.

It gives us pleasure also to acknowledge once again the generosity of the Medical Board. The grant for 1955 was £750, and with a credit balance of £75 5s. 6d. we started the year with £825 5s. 6d., which was spent as follows: books, £100 8s. 10d.; journals, year books and indices, £446 11s. 2d.; binding, £225 4s. 6d.; leaving a credit balance of £53 1s.

The Library Committee again would like to thank Miss Hagan, the librarian, for her excellent stewardship.

At the request of the University the present Library Committee has carried on during the new year and will continue to do so until June, when it is hoped that a new combined committee will take over office. As such, this will probably be the last report of the British Medical Association Library, which, like the phœnix, will rise again, having been reborn in the fires of the Central Medical Library.

REPORT OF THE POST-GRADUATE COMMITTEE.

The report of the Post-Graduate Committee was presented by Dr. Adrian Lamb and adopted. The report is as follows.

This committee of our Association exists to organize, correlate and stimulate post-graduate activities of all kinds in this State. Its members represent all the main channels of medical interest: practitioner, specialist, hospital, administrative, political and academic. In other States where there are medical schools this work is done by paid administrators; here it is done for fun. Last year your committee was felt to be unwieldy, and in an attempt to streamline it a small executive was appointed to meet monthly, whereas the whole committee would meet quarterly. This system has worked very well.

In 1954 this State was visited by a large number of medical eminences from overseas and from the remainder of Australia. This plethora of talent tended to exclude self-expression on the part of local workers, and this was vociferously pointed out by people who seldom came to any meetings anyway. To remedy this, during the past year almost the whole post-graduate programme revolved around men of this State who had something to say. In consequence we had a symposium on carcinoma of the cervix, a clinical evening presented by the Royal Perth Hospital, Professor Stephenson on "Medicine and Town Planning", orthopædic problems in general practice, a Fremantle clinical evening, the Sims Professor in Surgery, the medico-legal evening and a discussion on the *Workers' Compensation Act*.

These monthly clinical meetings are but the shop-front of the committee's work. Behind the scenes much other work has been done.

BRITISH MEDICAL ASSOCIATION (WESTERN AUSTRALIAN BRANCH).

Statement of Assets and Liabilities at December 31, 1955.

ASSETS.

	£ s. d.	£ s. d.	£ s. d.
Subscriptions in Arrears			245 14 0
Sundry Funds Overdrawn—Car Badge Account			19 5 1
Investments—			
Australasian Medical Publishing Company, Limited—			
Debentures "A"-"D"		345 0 0	
Debentures "E"		1717 10 0	
		2062 10 0	
Commonwealth Treasury Bonds		1410 0 0	
British Medical Agency Shares		9 0 0	
			3481 10 0
Cash at Bank of New South Wales			322 3 2
Fixed Assets—			
British Medical Agency British Medical Association House Loan Account		15,448 9 7	
Furniture and Fittings at January 1, 1955	958 14 10		
Since Purchased	74 15 0		
	1033 9 10		
Less Depreciation	51 13 6		
		981 16 4	
			16,430 5 11
			£20,498 18 2

LIABILITIES.

	£ s. d.	£ s. d.	£ s. d.
Subscriptions in Advance			39 18 0
Sundry Funds in Credit—			
Australasian Medical Publishing Company, Limited: Sale of Lists	3 5 0		
British Medical Association House: Members' Loans	11,580 0 0		
Building Fund	1001 7 0		
Entertainment Fund	8 8 3		
Library Donations	27 11 2		
Cyril Bryan Fund	188 8 1		
Harry Lucraft Fund	22 1 0		
C.R.U. Grant	26 0 2		
Medical Board Grant	53 1 0		
Medico-Legal Golf	4 13 0		
Nelson Bequest	17 7 1		
Post-Graduate Committee	255 14 0		
Post-Graduate Grant	324 10 1		
Publicity Fund	89 9 2		
		13,601 15 0	
Accumulated Reserve Account—			
Balance at January 1, 1955	5516 7 8		
Add Excess of Income over Expenditure	1340 17 6		
		6857 5 2	
			£20,498 18 2

We report that we have audited the accounts of the British Medical Association (Western Australian Branch) for the year ended December 31, 1955. In our opinion the accompanying balance sheet is properly drawn up and exhibits a true and correct view of the state of the Association's affairs at December 31, 1955, and the attached income and expenditure account is also properly drawn up and exhibits a true and correct view of the Association's affairs for the year. Both are in accord with the best of the information and explanations given to us, and as shown by the books of the Association.

(Signed) STOWE AND STOWE,
Auditors.

An anatomy course has been run as in the previous year, in spite of the peculiar arrangements concerning the disposal or availability of cadavers deriving from the *Anatomy Act* and the influence of the Adelaide Medical Faculty. This we hope will change with the establishment of a chair of anatomy here. It was not found possible to carry on a course in physiology up to Fellowship standard, as no suitable teacher was available—Dr. Crosby felt unable to spare the time. However, for some time a course in pathology has been run at Royal Perth Hospital under the ægis of certain general practitioners, and this was enlarged and extended.

Over 20 doctors at varying stages in their careers were recommended to the Director of Post-Graduate Studies in London, Sir Francis Fraser, and courses, accommodation and facilities were arranged for them. In a few cases ship's surgeon posts were obtained. These are often difficult to arrange due to very short notice being given by the shipping companies.

During the year members of the profession were invited by the committee from time to time to visit country centres, and this committee is grateful to them for the trouble and time spent on these visits. Several of the country districts are very keen and active and appreciate deeply the efforts of visiting lecturers.

Last year we recorded on tape some outstanding lectures, and these have been much sought after by outlying groups of doctors. The Melbourne committee is now doing the same thing.

One of the chief duties of this committee is to arrange far ahead for programmes designed to include overseas teachers who may be invited to Australia a year or two years in advance. This committee has already arranged details for the class A lecturer for this year, has arranged general dates for the equivalent lecturer for 1957, and has indicated its preference for the invited speaker for 1958.

To avoid clashes with other clinical and business meetings held throughout the year, it was arranged at the request of the honorary secretaries of the honorary staff of each hospital that a correlated plan of all clinical meetings for the year should be attempted. Last March this was done, and as a result on only one occasion during the year did clinical meetings clash—an improvement on previous years. In due course, no doubt, the Faculty of Medicine in conjunction with the British Medical Association Post-Graduate Committee will plan and direct all post-graduate activities.

The year just ended having been devoted to local talent, the committee realized that there would be the cry for outside star-rating this year. The task was made easier by the fact that an extraordinary number of high-powered overseas teachers wish to come here. As a result the programme for the coming year is full of variety and interest. Most of the clinical evenings are already arranged from a dermatological symposium by our own colleagues in April to a post-graduate week built round Professor Sheldon later on. The approximate programme will be published in the next issue of the British Medical Association Bulletin.

The future of this committee is in the melting pot in view of the establishment of the Medical Faculty, but it continues to organize the best post-graduate teaching it can, and I would like to say that any success it has had in the last year has been due to the able and pleasant inspiration of its chairman, Dr. Clement.

And finally, Sir, having formally resigned my stewardship to this committee, I am reminded of the story of an eminent Scottish physician playing at Saint Andrews. His caddie knew the course and every stroke, and never ceased to tell the doctor which club to use. Coming to the short hole, the eighth I think, the doctor took out a number 3 iron. "Yer spoon", said the caddie. "Not at all", said the doctor, "I shall use my number 3." Whereupon he struck the ball firmly and holed out in one. "There, you see", said the doctor. "Aye", said the caddie, "but ye'd have done it better wi' a spoon."

ADRIAN LAMB,
Honorary Secretary.

REPORT OF THE HONORARY TREASURER.

The Honorary Treasurer's report was presented by Dr. D. D. Keall and adopted. The report is as follows.

I am pleased to be able to report that the balance sheet for the past year shows that our finances are in a healthy state and that our income exceeded expenditure by £1340. Income was about £950 more than in the previous year, roughly half being due to the increase in British Medical Agency Company revenue. This latter increased from £280 to £820, and a substantial further increase is anticipated for next year.

Expenditure was over £1200 more than in 1954, the bulk of the increase being in salaries and the library.

During the year the Association's offices were transferred to its own building, and this is shown by a reduction of approximately £100 in the rent paid. In future, rent will not be a cost to the Association.

The purchase of Clarendon House has, so far, been an easy and painless proceeding, and we calculate that we have already acquired an equity of between £7000 and £8000 in the property. We all owe thanks to those who have subscribed to the building fund and to those who have made available interest-free loans.

Thanks to the honeyed tongue of his predecessor (I refer to Dr. Stubbe), who induced the members of this Branch to raise their subscriptions, the present treasurer's period of office has been remarkably free from anxiety. This pleasant state of affairs is about to be rudely changed now that the Council has decided to erect a building—at a very large cost—in front of the existing Clarendon House.

Dr. Copping will be reporting in detail on the proposed building, and it is my intention to confine my remarks to the financial aspects of the scheme. The plan is purely a business one; finding ourselves with suitable land and assured of suitable tenants, we are setting out, at an estimated cost of £65,000, to construct a block of professional suites, the rent from which will, after the building has been paid for, provide the Association with an annual income in the region of £6500.

The finance for the building will have to be borrowed, and the bulk of it will come either from a bank overdraft or a loan from an insurance company. If we are able to arrange for the former, provision will have to be made for repayment of the loan in ten years, and, in addition to the moneys from renting the suites, over £2000 *per annum* will have to be found from Association and British Medical Agency Company revenue. If this mode of finance (bank overdraft) is practicable, we shall be making a great drive for more interest-free loan money. These loans, large and small, for short or long periods (repayable on demand), are saving us considerable payments in interest, and their extension will make all the difference between our acquiring this new building in a hard or an easy way. So I appeal to any of you fortunate enough not to be in the red, to let us look after your surplus for you.

It is not for me to show how the Association should use the income which it will acquire if this venture is successful, but I feel it is not out of place to mention the possibilities of rooms and appointments in keeping with the dignity of our profession, various amenities and perhaps improvements in our arrangements for satisfactory relations with our profession and the public.

Last year I announced that it was Council's intention to ask a general meeting to reduce the annual subscription by £1 1s. The decision to embark on the new building made it impossible to implement this, as the moneys involved could make the difference between success and failure in financing the venture. Although the subscription cannot be reduced at present, we can expect, at a future time, both a reduction in our subscriptions and a substantial increase in the values received in return for them.

BUILDING COMMITTEE.

Dr. Copping presented a report from the Building Committee concerning the proposed erection of a building to house professional chambers at 8 Kings Park Road, which would become the headquarters of, and house the Association's offices, Council and committee rooms in future years. He referred to the methods at present being investigated by the committee to finance the project, which would be self-supporting. Dr. Crisp commented on the historical facts of previous building projects and suggested that it would be uneconomic to erect only a portion of the building now and add additional storeys later. Dr. Day referred to the advisability of acquiring two small blocks of ground immediately behind 8 Kings Park Road. Dr. Tresize suggested that the secretary should personally approach members for free-of-interest loans to assist the project. The president referred to the possibility of issuing debentures to add an additional floor for immediate British Medical Association use.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held in the Verco Theatre, Institute of Medical and Veterinary Science, Adelaide, on Thursday, October 27, 1955, DR. GRAHAM BENNETT, the President, in the chair.

The Prevention of Motor Accidents.

The meeting took the form of a symposium on the subject "The Prevention of Motor Accidents".

DR. GRAHAM BENNETT, from the chair, explained that those taking part would be a main speaker from the medical profession and three guest speakers from outside the profession. He said that the British Medical Association had always interested itself in measures for promoting public welfare, but in the past the attack on disease had been largely within the profession. In the future they would give increasing attention to aspects of disease and disability in which many social factors operated. Mental hygiene and adolescent delinquency, as well as the evening's subject, motor accidents, were examples. With their specialized training members of the medical profession were well fitted for understanding such human problems, and they could and should make an important contribution to the subjects mentioned. The symposium about to be presented was evidence of a determination to play a full part in the field concerned. They were indebted for the inspiration for the meeting to Professor J. B. Cleland, who had had great experience in the field with which he was to deal. He would be the main speaker. The guest speakers would be Senior Inspector J. J. Turnbull, Traffic Superintendent, Mr. F. Campbell, Secretary of the National Safety Council, and Mr. D. S. Dwyer, Touring Manager of the Royal Automobile Association of Australia.

PROFESSOR J. B. CLELAND said that public health was concerned with the physical well-being of the community at large. It was part of its duty to ascertain the causes of death and to endeavour to avert or reduce them. Among the most potent causes of death and disability at the present day were traffic accidents, mostly from motor vehicles. If the same degree of mortality and injury had been due to some disease which had recently greatly increased in incidence, many men with a medical training would be engaged in trying to control the disease and large sums of money would be employed in their investigations. Because the new danger was man made and not due to bacteria or a virus, the medical profession had not been explicitly called on to investigate the circumstances that led to motor accidents. As many members of the medical profession spent much of their time and mental energy in trying to elucidate the causes of illness—in the mass, as in public health, or individually, as by the family doctor—it seemed that they could play an important role in reducing the number of motor accidents if they were required as part of their duty seriously to consider the contributing causes. Medical men were more fitted for that than lawyers, the police or the person with a mission—though all those made valuable contributions—because of their special training in researches of such a nature combined with their knowledge of physiology, anatomy, mental reactions and psychology. Professor Cleland said that his suggestion to an important health authority that that body should engage in a study of the causes of motor fatalities had met with no response. Nevertheless, he put forward for consideration several ways in which he believed accidents could be reduced in number without undue restrictions being placed on motorists.

His first point was that every motorist should have his car under such control that he could avert an accident, however wrong the other person might be. He must be prepared for the other man's being intoxicated, not giving way on the right, swerving, and stopping without adequate notice. That would mean that, under the *Traffic Act*, it would be an offence to be involved in a motor accident. The penalty need not be great, though not negligible, and provision would have to be made for occasional cases in which no human care could have avoided the result—that would be decided by a magistrate. Thus no longer would the excuse be valid that it was the other person who should have given way or was in the wrong.

Professor Cleland's second point was in connexion with giving way to anyone coming in on the right. That, of course, was admirable up to a point. That the point was only half-way to a solution was shown by statistics supplied from time to time in the Press, which showed that "not giving way" was one of the commonest motor offences. That did not mean that most of the offenders should have known better. Many of them were doubtless not the ones to blame, the offenders being those who came in on the right who were engaged in proceeding across an intersection at much more than twenty-five miles an hour, or who were reckless enough to say that, however slow the other motorist was, it was his duty to let him go by. The explanation of the prosecutions was a very natural one. Often an overworked police officer was called on to make inquiries. Since one of the persons involved had been run into by a person coming in on his right, whatever the circumstances he could be quite successfully prosecuted. If he was convicted, and was not really to blame, the circumstances were most unfortunate for the community, as it had always been an axiom of British justice that innocent persons should not be punished, and a cherished heritage from Gilbert and Sullivan that the punishment should fit the crime. A person wrongfully convicted was a person with a rightful grudge against society. Professor Cleland said that he had been nearly run into over a crossing by a person going at probably fifty miles an hour—the road had been clear for a considerable distance before he had begun to cross slowly (he was free-wheeling, itself a danger). A relative on a motor cycle and sidecar, though he had practically stopped before crossing, had been run into by a motor-car, which had knocked his vehicle across the road and into a fence; that presented evidence of a force which must have been exercised by a vehicle about to travel over the intersection at much more than the limit of twenty-five miles an hour. He was punished, whereas he had taken adequate precaution and was proceeding slowly. His assailant (a good term in the present case) escaped even a reprimand. The second point was, of course, covered by the first point, but it should be made clear that giving way to those coming in on the right was, in spite of an expressed judicial opinion, a relative and not an absolute matter.

Professor Cleland's third point, to emphasize the fact that accidents must not happen, was that the drivers of all the vehicles concerned in an accident involving the death of a person or his detention in hospital for more than twenty-four hours should have their driving licences automatically cancelled for a month. In any case, such persons would be more or less "jittered", and their judgement likely to be at fault for some while after such an accident.

Professor Cleland went on to say that the compulsory wearing of crash helmets by all motor-cycle riders had been emphasized again in a subleader in the *British Medical Journal* for December 25, 1954 (page 1538). It was stated that its value was endorsed by the Committee on Road Safety in 1947 and in 1952 and by the Road Research Laboratory in 1949. The late Sir Hugh Cairns was quoted in the following words: "There can be little doubt that adoption of a crash helmet as standard wear by civilian motor-cyclists would result in considerable saving of life, working time, and the time of the hospitals." The cost of crash helmets in Britain was given as ranging from 47s. 6d. to 65s. An additional advantage, though not a medical one, would be that it would be more difficult for persons to steal parked motor-cycles and go for joy-rides, as they would have to provide themselves beforehand with crash helmets or, going without them, be quickly detected.

In an abstract of Barbara Preston's "Focus on Road Accidents" (1954) it was stated that of a total of 4000 to 5000 persons killed on the road each year in England and Wales, 46% were pedestrians, 23% motor cyclists (including passengers), 16% pedal cyclists and 15% occupants of motor vehicles. Of the pedestrians killed, 25% were under ten years old and 45% over sixty-five years old. Experiences in South Australia were very different. The children killed were few. It was only in later life that pedestrian deaths became numerous. The City Coroner had kindly supplied, through his officer, Sergeant A. C. Horsnell, the following figures for the ages of persons killed in motor accidents in Adelaide, Port Adelaide and the adjacent areas between November, 1953, and October 22, 1955: under ten years, two passengers and eight pedestrians; between ten and fifty years, 105 passengers and 13 pedestrians; over fifty years, 44 passengers and 49 pedestrians. The explanation of the large number of pedestrians killed in Britain was probably the number of towns with dense populations and with many children playing in the streets on account of the small size of the allotments on which their homes were built. Municipal rating in South Australia should encourage plenty of space to play around houses. Questions to be considered were whether children should be allowed to play in the streets, whether soap-box racing should be allowed on the roads, and how the aged could be protected.

Another point worth consideration was that anything tending to make people nervous—and consequently less capable of meeting an emergency—was to be deprecated. Alarmist notices, such as the suggestion that the sites of accidents should be marked on the roadway, instead of making the danger area safer, might actually render it less safe.

Professor Cleland said that he could see no good grounds for considering that special tests for new drivers would reduce motor accidents. An inexperienced driver almost invariably played safe. The danger lay in experienced drivers lacking judgement, being inattentive or taking risks. Dr. Johnson had said that sudden fits of inadvertency would surprise vigilance, and Hippocrates that life was short, experience fallacious and judgement difficult.

MR. J. J. TURNBULL, Traffic Superintendent, considering the subject from the point of view of the Police Department, said that road accidents were the concern of the whole community, and it was pleasing to find that the medical profession was interested enough in the high incidence to devise ways and means of combating the toll. If the same degree of mortality and injury was due to some other cause, whether disease, flood or bush fire, every effort would be made with men and money to combat the disaster. But because they were only road accidents, and occurred so often, they were dull and unimportant to most people. Yet the toll of road accidents mounted each year. In 1954 there had been 173 persons killed and 3926 injured from 3098 accidents in South Australia. In all there were over 24,500 accidents; so one could well imagine the cost of damage to vehicles apart from the waste of life.

Professor Cleland had made certain suggestions in his paper for the prevention of accidents. Whilst admitting that they had possibilities, Mr. Turnbull did not agree entirely with them. He agreed that every driver should have his vehicle under such control that he could reasonably avert an accident. In fact, the driver could be prosecuted for careless driving if it could be proved otherwise. At the same time, it was only reasonable for the driver to expect care and consideration from the other driver. Mr. Turnbull would not support any movement to prosecute a driver who was involved in an accident which was caused by another road user.

The second point related to "right of way". Failure to yield right of way was a major cause of road accidents, but one could hardly blame the law for that. In Mr. Turnbull's opinion it was purely the fault of the individual. Their figures had always shown that over 80% of road accidents were attributed to human failure, and that, with proper observance of road rules and care, the accident need not have occurred. True accidents were those due to a mechanical breakdown of the vehicle, but the rest were due to mistakes of human judgement, errors in driving or deliberate neglect. The right-of-way rule was very important for the guidance of drivers in general.

The third point in the paper related to penalty. Mr. Turnbull was quite satisfied that the courts would inflict an appropriate penalty on a careless driver responsible for death or injury in a road accident. But British justice would not be carried out if a driver was penalized for a death or injury when it could be proved that the other person was at fault; for instance, in 1954 20 pedestrians had been killed under the classification of "walking across roadway without due care". That was road suicide.

Mr. Turnbull went on to say that he doubted whether driving tests for new drivers would considerably reduce the accident rate. Their figures had always shown that it was the experienced driver who had the serious accident. The new inexperienced driver might have a trivial accident, but on the road he usually drove very carefully and slowly.

Mr. Turnbull did not agree with the compulsory wearing of crash helmets by motor-cycle riders, especially in the local hot summer conditions. It raised the question of whether pillion passengers should also be forced to wear one; they were just as vulnerable as the rider. The helmet undoubtedly would be a protection, but the wearing of it should be left to the wishes of the rider or passenger.

On the question of how to reduce the accident rate, Mr. Turnbull said that he was afraid he could not offer any suggestion other than that the individual road user must be educated to realize that there was a danger if he did not use courtesy, common sense and care. And furthermore he must be tolerant and obey all road rules. Laws were

made only after careful consideration; and no matter what laws were made or traffic facilities provided, the final result was in the hands of the road user. He could do the right thing when a patrol policeman was near by—why not when there was no policeman? Self-discipline did not need a policeman at one's elbow all the time. If the road user could not be educated, then the law must be enforced. But he did not consider that a harsh enforcement was the answer. It was notorious that in the past the criminal law of England had been harsh in the extreme; yet that did not abolish crime. What was required was a better understanding, brought about by education, that any antisocial conduct led only to an unhappy existence for all. It was necessary to build up an attitude of mind in the individual which would make him control his actions and realize his responsibilities; when that was attained, the accidents would be reduced. Mr. Turnbull believed that the family doctor could help considerably in that education. He could take the message to all members in the household, and no one was better qualified than he to understand the human mind. By doing so, he would be assisting in stamping out a blot on the intelligent existence of man.

MR. F. CAMPBELL, Secretary of the National Safety Council, said that the first point raised by Professor Cleland was that every motorist should have his car under such control that he could avert an accident, however wrong the other person might be. To impel him to exercise such control, provisions under the *Road Traffic Act* would make it an offence to be involved in an accident. That, to Mr. Campbell's way of thinking, would be both logical and sound in principle; but he was of the opinion that they had a long way to go before that could be brought about.

In the first place he thought that they had to be more fair to the motorist, and by the term "they" he meant, in particular, the legislator and the educationist. In that regard he was of the opinion that the paramount need was for road traffic laws and regulations to be adequate, yet simple in the extreme, and capable of easy interpretation. Often he had received telephone calls from motorists asking him to define the "right-of-way rule" at the various intersections within the metropolitan area. And often also it took him several minutes to explain in detail the complexities of each situation. He put the question whether it was, therefore, fair to expect the average motorist to be sufficiently aware of those particular intricacies of the law to enable him to make up his mind in a split second on the correct procedure to be adopted. Even if it was considered impracticable to simplify the present Act and regulations, he wondered if it would not be possible for a simple code of road ethics to be issued to all road users. That, of course, was where the educationist also came into the picture.

With regard to the second point raised by Professor Cleland, Mr. Campbell rather felt that the absolute right of way to be accorded to anyone coming in on the right was not really as absolute as it would sometimes appear in the courts; nevertheless, it might ease the minds of the motoring public if that particular section of the Act relating to the right of way could be construed more liberally by the public and legal profession alike, to indicate prima-facie evidence of negligence only, in cases of accident in which it was alleged that there was failure on the part of one party to give the right of way to another.

Mr. Campbell agreed with the third point raised by Professor Cleland and was of the opinion that motor vehicle drivers concerned in an accident involving the death of a person should have their driving licences automatically cancelled for one month. That condition, attached to the granting of a driving licence, would, he felt, be accepted by the motoring public of the State.

With regard to markings on roadways referred to by Professor Cleland, the National Safety Council did not favour them, mainly because accidents had been known to be caused through their distracting the attention of motor vehicle drivers.

As a keen advocate of driving tests, Mr. Campbell said that, whilst he agreed with Professor Cleland that danger lay in experienced drivers' lacking judgement, being inattentive or taking risks, he did think that many accidents occurred which could be avoided if the drivers involved had had a fairly rigorous training to fit them for the busy highways of the present day. A course for all motorists in first aid, too, would be of great value and could perhaps be included as part of the driving tests to follow.

On the subject of crash helmets (safety helmets was perhaps a more apposite term), the Australian Road Safety Council, of which the National Safety Council was a constituent body, had cause to be grateful to the medical profession for statistical and other data supplied on the nature of injuries sustained by motor cyclists; and it would be of some interest to those present at the meeting to know that the Council did in fact desire to encourage the motor cyclist voluntarily to wear a safety helmet.

The National Safety Council was also ever aware of the practical contribution the medical profession had rendered from time immemorial to the saving of lives and limbs of accident victims. In the prevention of road accidents they had contributed much. He would refer particularly to the scientific proof formulated to determine the degree of intoxication of a person "under the influence"; that, he understood, when related to the road accident problem, served to exonerate the innocent party, whilst at the same time it laid the blame on the guilty party.

Following that trend of thought, perhaps the Association could consider the setting up of a road accident research section to provide the legislator and enforcing authorities, the engineer and the educationist with some material aspect of road accident prevention based on (to use Professor Cleland's words) physiology, anatomy, mental reactions and psychology.

Mr. Campbell said that he put that point having regard to the road accident statistics, which were classified under "causes" such as "speeding", "inattentive riding or driving", "failing to give way to the man on the right" and so on; he often wondered whether enough was known about the underlying causes of those "causes". One might take "speed" for just one example. It was agreed that that was the cause of the accident; but the vital scientific and medical information was missing—what made the motorist speed?

One might also consider some of the youngsters who attended speedway meetings. Something happened to them during those meetings which seemed to exhilarate them, with the result that, on leaving the enclosure, they became somewhat inebriated with the exuberance of their own velocity—sometimes with the most disastrous results. Perhaps medical research would provide a pill that those young gentlemen could take to counteract the tendency.

MR. D. S. DWYER, Touring Manager of the Royal Automobile Association of South Australia, said that Professor Cleland struck a very important note when he classified road accidents as a public illness, for an illness it no doubt was. It behoved them, therefore, to prescribe the right medicine, and having once prescribed it, to take it in the right doses.

Most accidents on the road, like many diseases, were preventible; and as preventive medicine assisted the health of the community, so good driver behaviour assisted safety on the roads. As Professor Cleland had so clearly stated, accidents were "man-made" diseases, in that the mishap on the road was due in the main to one of the following causes: carelessness, aggressiveness, lack of courtesy, lack of consideration—or, in other words, failure to observe strictly the requirements of the *Road Traffic Act* tempered with common sense.

It seemed to Mr. Dwyer, when listening to Professor Cleland, that they must strike at the very root cause of the trouble in the same way as the medical profession set about curing the patient. Professor Cleland had prescribed a number of remedies as a means of reducing the accident rate. To accept the principle that it would be prima-facie evidence of an offence to be involved in a traffic accident would, Mr. Dwyer believed, be a negation of British justice. He envisaged a violent resistance by the majority of the people as an affront to their deeply ingrained sense of fair play. So much resentment and antagonism would be engendered that he questioned whether any government would touch such a proposal. If they assumed for a moment that it was in force, he wondered what would be the psychological reaction to a conviction under the *Road Traffic Act* followed by absolvement from blame by a civil court judgement under common law, as would certainly happen in hundreds of cases. The law would be regarded with contempt, and a conviction would be meaningless. There was no doubt the cure would be worse than the disease. Another aspect of the mass prosecution idea was that in 1954, 12,304 accidents had been reported in South Australia. *Ergo*, the police and courts would be swamped and hopelessly bogged down.

Mr. Dwyer agreed that there was a class of individual in the community who insisted on his legal right of way and pressed on regardless of the other fellow, showing complete disregard for courtesy and consideration.

With regard to the human element in relation to road behaviour, Mr. Dwyer said that he was very interested in an

article appearing in the *Autocar* of August 19, 1955, in which Dr. R. J. Smeed, of the Road Research Laboratory of London, commented: "That because a motorist should adjust himself to dangerous conditions (by, say, slowing for a bad surface) the guilt does lie on his shoulders in many cases if he does not; human nature being far from perfect, however, the driver does not adapt himself to meet dangerous conditions, and the result is the high accident rate prevailing today." He went on to say: "We cannot change human beings, although we can bring about improvement by education, as well as alter road layout and improve surfaces to reduce the possibility of accidents. Therefore it is no good saying that the driver should have slowed for an inferior road surface when he had skidded on it; the dressing on top of the road should have been such that he could not have locked his wheels, even if conditions were bad."

Mr. Dwyer said that, generally speaking, the whole of the traffic problems and accidents were bound up in the three "E's"—education, enforcement and engineering. The first-named with all its ramifications covered those important aspects "courtesy" and "consideration", while "enforcement" was the method by which the police controlled driver behaviour on the roads, by virtue of the *Road Traffic Act et cetera*. Naturally, "enforcement" must be tempered with British justice, and he had already given his reasons for regarding Professor Cleland's proposal as a negation of British justice.

Finally, "engineering" was the method of providing for the proper and safe passage of motor vehicles over the roads. It was in that direction that so much could be done in the field of traffic engineering, particularly that which covered improvement to bad corners and dangerous intersections and traffic-flow channelization by means of traffic islands and like road furnishings. It was in that work that the Association especially directed its energies by conferring with the Highways Department, local governing bodies, progress associations, school committees *et cetera*. They not infrequently received very valuable suggestions from members of the medical profession, who had submitted to the Council thoughts and ideas which had resulted in improvement to particularly dangerous localities. The Association at all times welcomed any thoughts and ideas which were directed towards road improvement and road safety.

Mr. Dwyer went on to say that shortly after his opening remarks Professor Cleland had stated that members of the medical profession were ideally equipped, owing to their scientific background and training, to assist in research on accident causation. There was no doubt that the prevention of accidents was a national problem, and as such all should lend their weight in improving safety on the roads; and he certainly agreed with Professor Cleland that the profession's contribution could be great. That, of course, provoked the question of how the profession could contribute. He would like to furnish a possible answer, although he was speaking without the authority of the National Safety Council. As in the Royal Automobile Association, where they had a member of the medical profession, a lawyer, engineers, a chartered accountant, pastoralists and so on, on the Council of the Association, so had the Traffic Committee of the National Safety Council an engineer, a lighting engineer, an insurance manager, a representative from the army, a senior police officer, and a representative from the Royal Automobile Association; but there was no member of the medical profession on that important committee, whose function was to handle the traffic safety side of the Council's activities. He left them with that thought.

He said that he would also like to comment on the matter of driving tests, which were apparently not favoured by Professor Cleland. The Royal Automobile Association supported those tests in the conviction that they started the new driver on the right foot. At least the applicant got his licence only when the examiner was satisfied that any bad habits which might have been copied from others had been eliminated, and generally he was up to standard to drive on the roads. It was realized, of course, that once a driver had been tested, bad habits could develop later in some; but that, of course, must be accepted, human nature being what it was. However, it did not detract from the value of driving tests as such.

There was no doubt that in the final analysis, road accidents were due in the main to individual road behaviour. It was for that reason that every motorist should carry out a "stocktaking" of just what he did by asking himself the following questions; if his answer was "yes" to each one, the result would be no accidents on the roads: "Do I always stop at 'stop' signs?"; "Do I always give proper hand signals?"; "Am I especially alert when aproaching an intersection or junction?"; "Do I pass cars only when it is safe to do so and overtake at intersections?"; "Do I observe speed limits and highway signs?"; "Do I drive with courtesy, care and attention all the time?".

DR. C. C. JUNGFER, in opening the discussion, said that Professor Cleland's paper was a challenge to the medical profession. The meeting showed that the British Medical Association, whilst accepting the challenge, was also aware of the need for coordinating its efforts with those of other bodies interested in the problem. One problem on which doctors could continue to work was the vexed one of human behaviour. Although that contributed to most motor accidents, it was not a new one, as was shown by the following extract given to Dr. Jungfer by Mr. Richmond, Commissioner of Highways, from the *South Australian Advertiser* of June 23, 1863:

> Woodside, June 19th. From our correspondent. Last Tuesday night, about 10 o'clock, as three of our neighbours, Messrs. Tonkin, Sen., H. Tonkin and Lewis, were on their way home from Adelaide, in a spring cart, and about a quarter of a mile from Oakbank, a German waggon was being driven very fast in an opposite direction, and the night being dark a fearful collision took place which resulted in the complete overturn of both cart and horse, hurling all three of the persons before-mentioned to earth. Mr. Lewis was for a time under the horse, and has broken his collar bone, besides being much bruised in other parts of the body. Mr. Tonkin and his son had a severe shaking but were not much hurt. The person who was driving the waggon did not think it worth while to stop, but seemed to increase the speed of his horse, and aided by the darkness of the night, has eluded all possibility of detection.

Dr. Jungfer said that human nature had not altered in ninety years, but the weapons had become more lethal. The medical profession should devote attention to recording and studying the human factors involved in motor accidents. They were as important as the studies made by lawyers and engineers.

DR. J. L. STOKES pointed out the importance of vehicular design in the prevention of serious injury in vehicular accident. He mentioned the present lethal design of the steering column, dashboard and door handles. He also stressed the great human problem involved in accident prevention, and recommended that high quality radio propaganda be used much more extensively.

DR. C. M. DELAND said that there were two colours of car which were dangerous—black at night and grey during the day, especially on a country road. Manufacturers should be encouraged to produce cars of more suitable colours. Regarding driving tests, he thought that they usually contained too many trick questions to be of real value. To overcome the problem of the ineffectiveness of a second injection of antitetanic serum, he suggested that all phials should be labelled: "The person receiving this should be actively immunized with tetanus toxoid."

DR. N. J. BONNIN wondered whether certain basic information necessary for the study of the problem was readily available—for example, if there was a register of accident-prone people.

DR. A. C. MCEACHERN contrasted the Australian preoccupation with rules, regulations and punishment with the English Highway Code, in which the emphasis was on care, prevention and good manners.

DR. J. L. STEELE SCOTT suggested that accident-prone drivers should have a lower speed limit, and that motorcyclists who wished to travel over 30 miles per hour should be made to wear crash helmets.

DR. O. W. LEITCH wondered what a doctor should do when (*a*) he found that a person with a driving licence was unfit to drive for medical reasons and (*b*) a person "under the influence" was admitted to hospital after a vehicular accident.

DR. P. S. WOODRUFF compared the Australian laws regarding "driving under the influence" and the strict enforcement of severe penalties in some other countries such as Sweden.

DR. A. R. SOUTHWOOD pointed out that there were more deaths as a result of vehicular accidents than of infectious diseases. He considered the best approach to be through the education of young people before their driving habits became fixed.

DR. J. M. DWYER emphasized the importance of vehicular design and the use of crash helmets.

DR. J. BONNIN favoured the development of three-lane roads.

DR. C. DUGUID suggested that motor vehicles be designed to have a limited top speed.

Professor Cleland, in reply, hoped that the medical profession could do something to help reduce the road toll. It needed a man with sufficient time to do research in the subject, and he suggested that an officer of the Department of Health be made available.

Inspector Turnbull, in reply, to Dr. N. Bonnin said that insurance companies had lists of accident-prone people. To Dr. Leitch, he said that doctors need only communicate with the police regarding drunks and medically unfit people. He went on to say that it was interesting that South Australia had the highest vehicles-population ratio of all the States and yet had the second lowest accident rate in the world. He therefore doubted the value of driving tests. Concerning three-lane roads, they had been shown to be very dangerous because of the fact that moving traffic on each side approached almost head-on.

Mr. Campbell pointed out the high cost of propaganda over the radio. He stated that the National Safety Council would welcome medical representation on their Traffic Committee.

Mr. Dwyer agreed with the value of statistics—for example, concerning accident-prone drivers.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE STATE OF A HOSPITAL IN 1803.[1]

[From the Wentworth Papers.]

Parramatta,
1 January, 1803.

Sir,

I send you the Returns for the two last months enclosed, by them you will see that there has been an unusual number of deaths here and in the neighbourhood, and from the number of patients now in the hospital ill with dysentery and other diseases, together with the deplorable State of the Hospital itself, I must request that you will represent to his Excellency, how necessary it is that the Hospital should have a complete and speedy repair. It is really impossible for me to describe to you the wretched State of the Patients as to Bedding, which there is almost a total want of, nor have I had a single grain of either Oatmeal Barley or Sago for some time past. I beg you will order me the necessary and medicines in the enclosed list being very much in want of them and

I am, Sir,

Your most obedient Servant,

D. WENTWORTH,
Staff Surgeon.

Thos Jamison Esq.,
Acting Surgeon General.

[1] From the original in the Mitchell Library, Sydney.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Revision Course at Rachel Forster Hospital for Women and Children.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a full-time revision course will be held from Monday to Friday, July 23 to 27, 1956, at the Rachel Forster Hospital for Women and Children, Redfern. The course is open to all members of the medical profession, and limited accommodation is available for women graduates at £4 4s. The programme is as follows.

Monday, July 23: 9 a.m., official opening by Sir Charles Bickerton Blackburn; 10.15 a.m., demonstration of leucorrhœa, punch biopsies and smears of cervix, Dr. Gwen Downes; 11.30 a.m., demonstration of pre-operative gynæcological patients, Dr. Gwen Gerrard; 2 p.m., "The Anæmias", Dr. R. Walker; 3.15 p.m., Arthritis Clinic—demonstration of cases and discussion of treatment, Dr. Claire Weekes and Dr. Nanette de Coek.

Tuesday, July 24: 9.15 a.m., "Rheumatic Fever and Nephritis in Children", Dr. Elizabeth Miller; 10.30 a.m., "Heart Disease: Case Demonstrations and Discussion", Dr. Jean Palmer; 2.15 p.m., "Summary of the Surgical Attitude to Peptic Ulcer, Hiatus Hernia and Œsophageal Reflux", Dr. S. H. Lovell; 3 p.m., "Management of Minor Surgical Conditions", Dr. S. H. Lovell; 4.15 p.m., surgical panel (open question time), Dr. S. H. Lovell and Dr. A. W. Middleton.

Wednesday, July 25: 9.30 a.m., "Dermatological Therapy", Dr. Jean Mason-Johnson; 10.15 a.m., "Anæsthesia", Dr. Gwen Bernard; 11.15 a.m., ear nose and throat: (*a*) "Treatment of Some Associated Respiratory Tract Infections", (*b*) "Otitis Externa: Observations on Some Causes and Their Treatment", Dr. Brenda Laws; 12.15 p.m., "Vertigo and Deafness", Dr. Patricia Davey; afternoon: medical ward rounds.

Thursday, July 26: 9.30 a.m., "New Techniques in Pathology", Dr. Jean Drury-White; 10.15 a.m., "The Early Diagnosis and Types of Cerebral Palsy", Dr. Claudia Burton Bradley; 11 a.m., demonstration of the stages of learning to walk by spastic and athetoid children, Dr. Claudia Burton Bradley; 11.45 a.m., "Modern Uses of Blood and Blood Products", Dr. R. J. Walsh; 2 p.m., demonstration of cases of carcinoma and other breast conditions, Dr. Kathleen Cuningham; 4 p.m., "Fluid and Electrolytic Balance in Surgical Patients", Dr. Margery Scott-Young.

Friday, July 27: 9.15 a.m., new drugs: (*a*) "New Drugs in Psychiatry", Dr. Joan Fotheringhame, (*b*) "Antibiotics", Dr. Helen Taylor, (*c*) "Peripheral Vasodilators", Dr. Pat Manuel, (*d*) "New Drugs in the Treatment of Epilepsy and Parkinson's Disease", Dr. Gwyneth Roberts; 10.45 a.m., "Practical Points in the Management of Diabetes Mellitus", Dr. Willa Nelson; 11.15 a.m., "Clinico-Pathological Conference", Dr. Helen Taylor; 2 p.m., "Investigation and Treatment of Infertility", Dr. Del Pufiett.

The fee for attendance is £3 3s., and written application, enclosing remittance, and indicating whether accommodation is required, should be made to the Course Secretary, Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, not later than June 29, 1956. Telephones: BU 4497, BU 4498. Telegraphic address: "Postgrad Sydney."

Course in Advanced Surgery.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a course in advanced surgery, suitable for candidates for the final F.R.A.C.S. examination and the final F.R.C.S. (England) examination, will be held for three months beginning on July 23, 1956. This course will conclude prior to the final F.R.A.C.S. examination in general surgery, which will begin in Sydney on October 19, 1956.

The course will consist of afternoon demonstrations, which will take place at the Royal Prince Alfred Hospital, Sydney Hospital, Saint Vincent's Hospital, Royal North Shore Hospital and the Royal Alexandra Hospital for Children. During the final month, candidates who are able may attend morning lectures to be held in the Post-Graduate Theatrette at 131 Macquarie Street, Sydney. The demonstrations held at the teaching hospitals will be case demonstrations including surgical pathology and management of operative technique, and post-graduates attending this course will be given frequent practice in the examination of patients, pathological specimens and X-ray films under examination conditions.

The fee for attendance is 35 guineas, and application should be made to the Course Secretary, Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney, from whom a detailed programme may be obtained. Applications close on July 16, 1956. Telephones: BU 4497, BU 4498.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 22, of May 10, 1956.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Southern Command.

Royal Australian Army Medical Corps (Medical).—3/101020 Captain (provisionally) D. N. Hawkins ceases to be seconded whilst undergoing post-graduate studies in the United Kingdom, 13th March, 1956. 3/101830 Captain (provisionally) A. J. Rollo relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command) in the honorary rank of Captain, 6th November, 1955. *To be Captain (provisionally), 23rd March,* 1956.—3/101836 Graeme Roderick McLeish.

Tasmania Command.

Royal Australian Army Medical Corps (Medical).—3/129187 Captain (provisionally) P. H. G. MacCallum relinquishes the provisional rank of Captain, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Tasmania Command) and is granted the honorary rank of Captain, 30th December, 1955.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

Northern Command.—To be Honorary Captains—Henry Andrew Muir, 23rd March, 1956, and Keith Edgar Free and Thomas John Thatcher, 26th March, 1956.

Eastern Command.—To be Honorary Captain, 23rd March, 1956—Alfred Brian Corrigan.

Retired List.

Eastern Command.

The name of Lieutenant H. A. Muir is removed from the Retired List on appointment to the Reserve Citizen Military Forces, Royal Australian Army Medical Corps (Medical) (Northern Command), 23rd March, 1956.—(Ex. Min. No. 57—Approved 3rd May, 1956.)

Research.

EXPERIMENTAL RESEARCH INTO PROBLEMS OF AGING.

THE trustees of the Ciba Foundation for the Promotion of International Cooperation in Medical and Chemical Research, 41 Portland Place, London, W.1, wishing to encourage well-conceived research relevant to basic problems of aging, invite candidates to submit papers descriptive of work in the field for awards for 1957. Copies of the regulations and form of application must be obtained from G. E. W. Wolstenholme, Director, and Secretary to the Executive Council, before an entry is submitted, but in general candidates should note the following: (*a*) Not less than five awards, of an average value of £300 each, are available for 1957. The announcement of awards will be made in July, 1957. (*b*) Entries must be received not later than January 31, 1957. (*c*) Entries will be judged by an international panel of distinguished scientists including Professor C. H. Best (Toronto), Dr. E. Braun-Menendez (Buenos Aires), Professor E. J. Conway (Dublin), Professor G. W. Corner (New York), Professor A. Haddow (London), Professor V. R. Khanolkar (Bombay), Professor R. Nicolaysen (Oslo), Dr. A. S. Parkes (London), Professor F. Verzár (Basle), and Professor F. G. Young (Cambridge). They will advise the Executive Council of the Foundation on their findings and will also have power to recommend variation in the size and number of the awards according to the standard of entries. The decisions of the Executive Council will be final. (*d*) In making the awards preference will be given to younger workers. (*e*) The work submitted should not have been published before May 31, 1956, though it may be under consideration for publication at that date. (*f*) The papers may be in the candidate's own language. Papers should not be more than 7000 words in length, and in all cases a summary in English must be attached not exceeding in words 3% of the length of the paper. (*g*) Where there is one or more co-author, the name of the leading author should be indicated; it is to him that the award will normally be made, and it will be left to his discretion to share this award appropriately with his co-authors.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 19, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	4(4)	3(3)	1(1)	..	1(1)	..	..	..	9
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1(1)	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1	16(14)	2(2)	..	1(1)	..	..	..	20
Diphtheria	2	4(4)	..	..	5(4)	..	..	..	11
Dysentery (Bacillary)	..	5(1)	6(6)	1(1)	6(5)	..	..	..	18
Encephalitis	1	..	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1(1)	..	..	..	..	..	..	1
Infective Hepatitis	83(38)	63(29)	..	10(1)	6(4)	4	..	..	166
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	4	..	4
Leptospirosis	1	..	11(1)	..	..	..	..	..	12
Malaria	..	1(1)	3(3)	..	..	..	3	..	7
Meningococcal Infection	4(1)	..	..	..	..	..	..	..	4
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	8(6)	6(3)	5(4)	..	1(1)	1	..	..	21
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	9(3)	..	2(1)	..	..	..	..	11
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	22(10)	17(10)	3(1)	1	2(2)	..	..	..	45
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1	1	..	1	..	..	..	3
Trachoma	..	..	..	..	8	..	12	..	20
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	17(7)	18(7)	37(27)	12(8)	13(9)	5(2)	1	..	103
Typhoid Fever	..	2(2)	2	..	..	..	..	..	4
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

Figures in parentheses are those for the metropolitan area.

Honours.

BIRTHDAY HONOURS.

HER MAJESTY THE QUEEN has been pleased to confer the following honours on Australian medical practitioners.

Dr. Kenneth William Starr has been created a Companion of the Most Distinguished Order of Saint Michael and Saint George.

Dr. Leonard James Ternouth Pellew has been created a Commander of the Most Excellent Order of the British Empire.

Dr. John Matthew Dwyer has been created an Officer of the Most Excellent Order of the British Empire.

The College of Radiologists of Australasia.

EXAMINATION FOR FELLOWSHIP OF THE FACULTY OF RADIOLOGISTS, LONDON.

AN examination for the Fellowship of the Faculty of Radiologists, London, will be held in Sydney and possibly New Zealand early in May, 1957. The examination will be conducted by the Faculty of Radiologists in association with The College of Radiologists of Australasia, and will consist of examinations in medicine, surgery, pathology and either radiodiagnosis or radiotherapy. Any prospective candidates are urged to communicate with the Honorary Secretary of The College of Radiologists of Australasia, 135 Macquarie Street, Sydney, as soon as possible.

Notice.

THE AUSTRALASIAN ASSOCIATION OF PSYCHIATRISTS.

Course in Psychiatry, 1956.

THE Australasian Association of Psychiatrists will conduct the following lectures at 8.15 p.m. on the dates shown at 45 Spring Street, Melbourne, C.1, Victoria: July 10 and 12, Dr. John Williams, "Significance of Behaviour Problems in Children"; July 17 and 19, Dr. Alan Jeffrey, "Theory and Psycho-Pathology"; July 24 and 26, Dr. Ainslie Meares, "The Dynamics of the Interview"; July 31, Dr. Frank Graham, "Transference"; August 2, Dr. Frank Graham, "Analytical Group Therapy"; August 7, Professor Richard Lovell, "Recent Studies of Adrenal Cortical Function"; August 9, Dr. Doig, "The Experimental Approach in Psychosomatic Disorders"; August 14, Dr. Alan Stoller, "Brain Damage and Psychiatry"; August 16, Dr. Alan Stoller, "So-Called Functional and Organic Psychiatric Disorders".

The fee for this course is £10 10s., or £1 1s. for a single lecture. Enrolments should be made through the Melbourne Medical Post-Graduate Committee (394 Albert Street, East Melbourne, FB 2547), on their special form, and will be received until June 25, 1956.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Powell, Philip Arthur McSharry, M.B., B.S., 1953 (Univ Sydney), Bartasch Avenue, Henty.

Bromhead, Brian Charles, M.B., B.S., 1953 (Univ. Sydney), 18 Manuka Arcade, Manuka, A.C.T.

Medical Appointments.

Dr. L. V. Perrett has been appointed senior radiological registrar at the Royal Adelaide Hospital.

Dr. G. A. Hunter has been appointed medical registrar at the Royal Adelaide Hospital.

Dr. Paul Kelleher Smith has been appointed Quarantine Officer, Mackay, Queensland, pursuant to the provisions of the *Quarantine Act*, 1908-1950.

Dr. T. J. Ashton has been appointed Medical Registrar at the Royal Adelaide Hospital.

Deaths.

THE following death has been announced:

KIRKWOOD.—William Love Kirkwood, on May 24, 1956, at Epping, New South Wales.

Diary for the Month.

JUNE 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 18.—Victorian Branch, B.M.A.: Finance Subcommittee.
JUNE 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 20.—Victorian Branch, B.M.A.: Country Branch Meeting.
JUNE 20.—Western Australian Branch, B.M.A.: General Meeting.
JUNE 21.—South Australian Branch, B.M.A.: Scientific Meeting.
JUNE 21.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.